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CLINIC OF DR. ARTHUR R. ELLIOTT

St. Luke's Hospital

GALLOP RHYTHM

GENTLEMEN: I wish to talk to you today about a form of heart rhythm known as "gallop rhythm." This is the term ordinarily employed in referring to this phenomenon, although we encounter in the literature of the subject other designations, such as "triple rhythm," "three-quarter rhythm," and "third heart sound." The English call it "canter rhythm" and the French, "bruit de galop," and sometimes "rhythmus Brightique," because of its occurrence in Bright's disease. I am encouraged to ask your serious attention to the study of this symptom not only because of its inherent interest as a physiologic anomaly but also because of its very considerable prognostic significance when met with in certain conditions. I shall have 2 interesting illustrative cases to show you, but before doing so certain considerations should engage our attention. First of all, we must be careful to discriminate between the triple rhythm we are considering and the so-called splitting or reduplication of one or other of the heart sounds, which is perhaps the commonest change which we hear in the relative time occupied by the sounds and silences of the cardiac cycle. One or the other of the heart tones is replaced by two more or less clear and similar sounds. Split sounds are characterized by the absolute similarity of the two fragments and the exceedingly short interval between them. Although the ear is cognizant of three sounds, cardiac rhythm is not broken. The sounds do not follow each

other in three-four time, but the heart preserves the normal two-four time ($\smile\!\!\smile$ $\smile$). The double tones in splitting are repeated so rapidly and identically that the examiner gains the impression of but a single tone accented either at the beginning (*t*l*a* ta) or at the end (ta tat*l*). Splitting of the second heart sound is most common, and is heard best at the second or third interspace. There is still some. divergence of opinion as to what causes this diastolic reduplication at the base, but the explanation most current is that it is due to slight asynchronism of the two ventricles, or to the fact that even without this the pulmonic and aortic valves may not close at exactly the same instant-because of a difference of pressure within the two arterial circuits. Splitting of the second sound may occur in the normal heart at the end of inspiration, especially under circumstances of respiratory stress. In pathologic conditions it may have its origin in any state which disturbs the normal balance of the circulation, whether such arises in the heart itself or is extra-cardiac. Splitting or reduplication of the first heart sound is much less commonly met with. It is probably produced by a slight asynchronism between the two ventricles, so that the maximum intensity of their contraction fails to absolutely coincide. Systolic doubling is ordinarily best heard at the apex and over the body of the heart.

It will be seen that splitting of the heart tones does not possess any fixed or necessarily important significance. The phenomenon may arise under any circumstance that disturbs or hampers the smooth working of the cardiac mechanism. It may be very transient in duration or may last for long periods without appearing in the slightest degree to embarrass the heart in its function or to be a symptom of any important circulatory disease. It is to be clearly understood that a split but normal rhythm of the heart is a very different thing from what we are considering as triple or gallop rhythm. In this form of disturbed rhythm there is a third sound interpolated, so that the heart, instead of beating as normally in two-four rhythm, beats in three-four rhythm. The interpolated sound has been described as appearing in different associations in time

with regard to the normal sounds. This is by no means easy to detect, the ear rarely being capable of deciding which is the true systole and which the added or third sound. Whether the interpolated sound is presystolic, protodiastolic, or middiastolic, the resultant effect upon the ear is the same, and by agreement the general term "presystolic gallop rhythm" has been adopted, the acoustic effect resembling the sound of a horse galloping over a firm surface. When present, this triple rhythm is heard more or less distinctly over the entire heart. A slight variation in accentuation may be apparent in certain instances, the second of the three sounds being accented toward the apex ($\smile \; \smile' \; \smile \quad \smile \; \smile' \; \smile$), whereas the accent may appear to shift somewhat to the third sound at the base of the heart ($\smile \; \smile \; \smile' \quad \smile \; \smile \; \smile'$). The area of audibility is especially the mitral and tricuspid regions; it is practically never heard with maximum intensity over the base of the heart. In occasional instances we may see or feel, in addition to the normal systolic impulse, a diastolic or presystolic shock or wave on the anterior chest wall corresponding to the third heart sound. There is never duplication of the arterial pulse, although a certain indescribable alteration of pulse rhythm may enable the experienced finger to detect gallop rhythm at the wrist.

The explanation of this peculiar rhythm is not yet entirely clear. Clinical observation would appear to indicate that it is apt to occur whenever the muscular tonus of the ventricles is impaired either from the effects of myocardial disease or as a consequence of functional overstrain. The former class of cases includes the failing heart of nephritis and high-pressure states, generally chronic anemias, adherent pericardium, etc., while examples of the latter occurrence are met with among athletes during trials of strength and speed, and not infrequently gallop rhythm may appear as a transient development during the febrile stage of acute infections, such as pneumonia, influenza, sepsis, etc. Potain's explanation of its pathogenesis is the most widely current. According to this authority the interpolated sound results from tension communicated to the wall of the ventricle by the entry of blood from the auricle during diastole.

It is more marked, according as the ventricular wall is less extensible, whenever the muscular tonus of the ventricles is impaired, so that the elastic resistance of the heart wall is in excess of its contractility. Under such circumstances it responds more readily to the shock of communicated tension, which tension is occasioned in the first place by the *vis-a-tergo* of the entering blood, and in the second place by the auricular contraction. Such an explanation lacks somewhat in simplicity, and from the clinical standpoint does not appear to fully fit the facts, for conditions which are directly opposed in nature produce gallop rhythm. Thus we meet with it clinically in such widely differing conditions as the stimulated cardiac action of health, the cardiac irritability of the toxic weak heart in pneumonia, influenza, typhoid, diphtheria, etc., the irritable heart of toxic goiter, and in the heavily beating hypertrophied heart of high blood-pressure states. Any explanation must take into account that the heart showing gallop rhythm is invariably a rapidly beating heart, and one that is either temporarily or permanently a fatigued and debilitated organ. It may be that Potain's theory fulfils this requirement, for it makes gallop rhythm the expression of an abnormally stimulated heart activity which produces either an excessively quick ventricular relaxation during diastole, with a consecutive sudden passive tension of the ventricular wall from the entering blood, either with or without an increased contraction of the auricle.

Whatever may have been the cause concerned in bringing about gallop rhythm, it serves as a sure sign that the heart is nearing the limits of its reserve. It is an indication of exhaustion; whether the heart be merely overstrained or is diseased, this is the significance of the sign, and its importance in prognosis is thereafter a matter of clinical interpretation. I have referred to its occurrence as the result of the cardiac overstrain of athletes. Trials of speed are more apt to cause gallop rhythm than are trials of endurance. Running and swimming races and bicycle races are the most apt to produce it. It goes without saying that sudden intensive physical effort will operate to produce gallop rhythm much more quickly in the mature and

the untrained individual than in the youthful and trained athlete. When produced as the result of sudden and severe functional overstrain it is usually associated with increase in cardiac diameter from dilatation, and often with jugular pulsation, indicating tricuspid insufficiency. This form of gallop rhythm is ordinarily of short duration, quickly disappearing with rest, and return of pulse-rate to normal. Acute febrile infections operate in much the same manner as do athletic overstrain to produce cardiac exhaustion from overstimulation, and similarly, we find gallop rhythm from infective toxemia a transient phenomenon coincident with the high pyrexial rapid pulse, and subsiding with decline in temperature and pulse-rate. It is perhaps most often heard during pneumonia as crisis approaches, and is an added unfavorable sign if the pulse is increasing steadily in frequency and the blood-pressure declining. It was often present during influenza and practically only in unfavorable cases. Rapid pulse and gallop rhythm make an unfavorable combination in diphtheria. The prolonged tachycardia of exophthalmic and toxic goiter, strange to say, does not often cause gallop rhythm, but febrile acute thyroidism usually will, and sooner or later it is observed in most cases of ulcerative endocarditis. The weak and rapid heart of the primary anemias will often gallop as the end approaches, and the persistent right heart overstrain of chronic asthma and emphysema often brings it about. It may be heard for weeks and sometimes for months over the heart in adhesive pericarditis, and is an unfavorable omen.

The foregoing states of cardiac exhaustion in which gallop rhythm is observed do not by any means complete the list; many other asthenic conditions in which it may develop as a terminal indication will occur on consideration.

I am particularly anxious today to enlist your interest in the association of gallop rhythm with the failing heart of high blood-pressure. This type of heart is a heavily beating, enlarged organ, having more in common with the athlete's heart than with any of the other states mentioned.

The ability of the greatly hypertrophied heart met with in

chronic nephritis and other high-pressure states to maintain an efficient circulation for long periods of time often covering many years is common knowledge. This type of heart seldom fails by acute dilatation. Failure is by a gradual exhaustion of myocardial tonus from persistent overstrain. As ventricular efficiency grows less, there is ordinarily a gradual progressive decline in systolic blood-pressure, with a coincident but not equally progressive drop in diastolic pressure. This double decline serves to maintain a sort of balance between the peripheral resistance and the heart power, and there is not much change in heart rate, the pulse remaining moderate, although the circulation is manifestly failing. This, then, is the ordinary type of heart failure occurring in high-pressure states. In certain cases high-pressure heart failure is characterized by different clinical manifestations and different behavior of pulse-rate and blood-pressure. I refer to the condition known as "high blood-pressure stasis." In these instances the pulse-rate becomes permanently rapid, gallop rhythm and alternating pulse often appear, and the blood-pressure, in place of falling, as one might expect, remains unaltered or may become still further elevated. This is particularly apt to happen on the diastolic side of the pressure formula, the highest diastolic pressures ever observed being in this condition. This high diastolic pressure represents the degree of obstruction at the periphery of the circulation, and in the face of such a block it does not require more than a small systolic output from a laboring ventricle to maintain an elevated systolic pressure—in other words, decline in the mass movement of the blood results and the circulation becomes increasingly static. The burden thrown upon the ventricle to maintain the circulation under these conditions impairs its contractility and apparently brings about the proper lack of balance between distention and contraction necessary for the development of gallop rhythm, for in practically all of these cases this rhythm is observed. In a series of 15 cases of high-pressure stasis which I recently reported gallop rhythm occurred in every instance. It may be well to point out that this rhythm of the ventricle is not due to

the absolute degree of blood-pressure, but to an aortic pressure relatively too great for the available myocardial energy. I will now show you 2 patients displaying presystolic gallop rhythm.

CASE I.—White male, aged fifty-five, who is now a resident of the hospital ward for the fourth time. He is a decidedly obese individual, weighing 200 pounds. He is a capmaker by occupation, is married, and has 5 living children. He was told five years ago that he had kidney trouble and a high blood-pressure, and came under my observation first in 1917, complaining of dyspnea ·and precordial distress following effort, and also he had early morning headaches which were markedly aggravated by a full diet, especially if he ate meats. He was rising thrice at night to urinate and had at times vertigo and palpitation. His past medical history was clear of any apparently significant illness or infection, and his habits had been moderate, with the single exception of eating, which he had done to excess. For ten years he had been aware of an intolerance for meats, yet had not abstained from them. His corpulence had steadily increased until the time he came under observation in 1917, when he weighed 235 pounds. His blood-pressure at the time was systolic 190, diastolic 130. Pulse 84 and regular. The heart was frankly enlarged and the second aortic sound was loudly accented, with an amphoric quality, as if the aorta were dilated. A moderate degree of emphysema was apparent. The liver was not enlarged, and although dyspnea followed effort, he was able to lie down comfortably. The urine was free from sugar, but contained a moderate amount of albumin, the sediment showing a few casts. Such was his condition more than three years ago. He remained under observation in the wards a fortnight, being bled twice (350 and 450 c.c.). During this period of observation his phthalein kidney function index was 40 per cent. for a two-hour period. A Mosenthal diet test demonstrated the fact that he was able to properly concentrate his urine. Spinal fluid Wassermann proved negative. He left the hospital considerably improved as to his headache and dyspnea and with a blood-pressure of

170/100. Gallop rhythm was not present during this observation. He remained in fair condition until October, 1918, when he experienced three attacks of angina pectoris, his blood-pressure at the time being 200/115. He entered the hospital then for a brief stay, and was bled once, with apparent benefit. His anginal attacks subsided and did not reappear severely until May, 1920, when he again entered the hospital. He was then found to have a blood-pressure of 235/140, gallop rhythm pulsus alternans, and much passive engorgement of the liver. There were many râles throughout the chest and nocturnal dyspnea made his nights extremely restless. There was no edema. The phthalein kidney index proved to be 40 per cent. and his blood urea nitrogen 17.04, with total non-protein nitrogen 37.11 mgm. Venesection to the extent of 750 c.c. was performed. Under the influence of rest, depletion, and digitalis his discomforts abated, gallop rhythm subsided, and he left the hospital well satisfied with his improvement. He did not long remain comfortable, however, any attempt at activity causing stenocardiac distress. Insomnia, cardiac asthma, abdominal distention, nausea, and anorexia made his life miserable until his last entry into the wards in September. It was now found that his pulse was persistently above 100, was alternating typically, although uneven as to rhythm, the heart galloped permanently, being no longer influenced by rest, digitalis, or venesection, which was done freely.

You will observe in how distressing a condition this poor man is. He sits bending forward the better to breathe. He has not been able to lie down for many days so great is his orthopnea, and he has much gravity edema of the feet in consequence. His abdomen is tympanitic and distended, but there is no demonstrable ascites. His respiration is rapid and labored and distinctly Cheyne-Stokes in type. He is depressed, at times mildly delirious.

His pulse now is never below 108 and the rhythm is irregular and always alternating. The blood-pressure averages about 200/130. I do not think I ever examined a heart that represented greater strain in action. The gallop is typical, and the

ringing tambourine note of the second sound can be heard by the unaided ear 2 feet from the chest. Blood nitrogen values have increased somewhat, urea being now 37.4 mgm. and non-protein nitrogen 62.32 mgm. The urine, of course, is albuminous and scant.

Electrocardiograph study of this patient shows a normal mechanism and normal conduction time, with occasional pre-
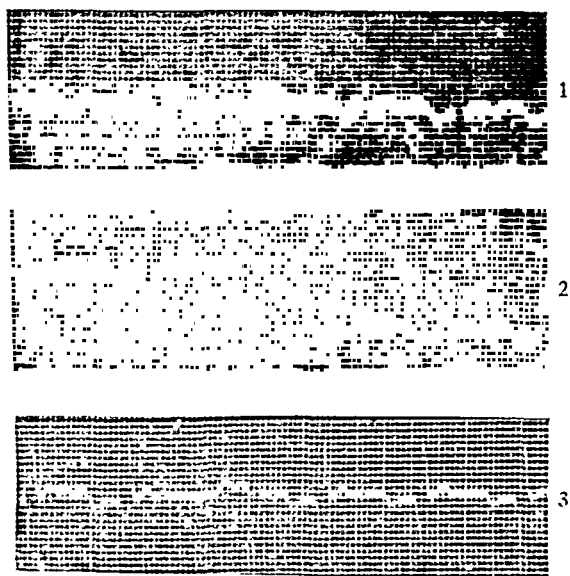
Fig. 1.—Normal heart mechanism; occasional premature beats of ventricular origin; negative T wave in all three leads (digitalis results); conduction time normal —16; R-S equals .08.

mature beats of ventricular origin. Negative T wave in all three leads probably results from digitalis, which has been administered. This brief review of a very interesting clinical history reveals a case of cardiovascular-renal degeneration with hypertension of some years' duration. The renal moiety of this combination does not represent an active nephritis, but a renal sclerosis, from which no important kidney insufficiency has developed. The moderate grade blood-nitrogen concentration

now present being little greater than might be expected from the kidney function decline of passive hyperemia. Despite the steadily advancing cardiac failure the blood-pressure has remained high, and it is especially to be noted that except for short periods following venesection the diastolic has not declined much below 130 mm. Six months ago gallop rhythm and alternating pulse developed and have persisted without interruption. It is obvious that the present state of affairs cannot long continue. With the inevitable antemortem drop in pressure we may expect gallop rhythm to disappear.

The foregoing is an extreme example of high-pressure stasis with gallop rhythm. Unfortunately, hospital ward material does not often furnish the examples seen more frequently in private practice of hypertension patients, who are ambulatory and possibly going about their business, and who have galloping hearts and even Cheyne-Stokes breathing, yet make little complaint of subjective distress except perhaps short-windedness and cough on exertion. It is in cases such as these that the significance of gallop rhythm counts in prognosis, for it may be said that a persistent gallop rhythm in a high blood-pressure case is a sign of the gravest import. Once established, it never wholly disappears, and the longevity outlook for the patient cannot be stretched beyond two or three years at the utmost, and usually weeks or months see the termination of the case. At times a patient with high blood-pressure will be seen who has a persistently rapid pulse, but whose heart does not gallop. One should take cognizance at once of tachycardia in high blood-pressure states. The normal pulse for an elevated pressure, other factors being excluded, is moderate or slow. A rapid pulse under these circumstances points to an insufficient ventricle. In such instances one of the usual effort tests employed to stimulate heart action may develop a latent gallop, and enable one to secure thereby a truer appreciation of how seriously the circulation is handicapped. The most striking examples of gallop rhythm are encountered in Bright's disease. I have such a patient to show you today.

CASE II.—This patient, a single man aged forty-five, normal

weight 240 pounds, came under my observation in private practice in September, 1920, being referred for examination because of high blood-pressure, which had been first detected at life insurance examination one month previously. His subjective complaints consisted of dyspnea on exertion and nocturnal urination. I shall pass over all considerations appertaining to his personal and family history, confining myself solely to such
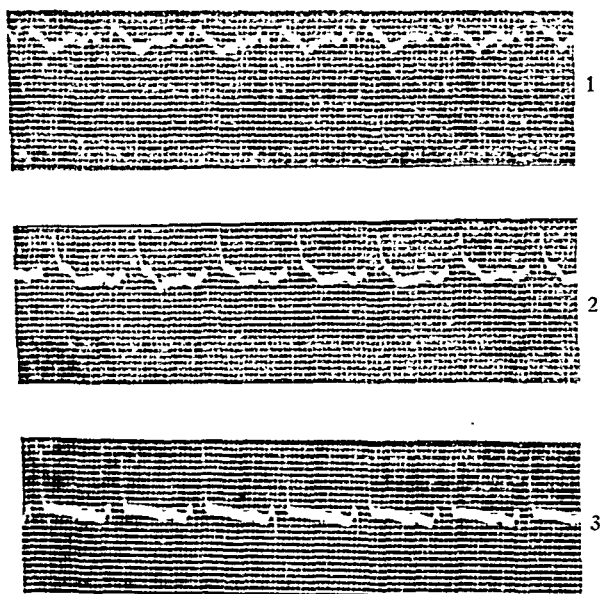
Fig. 2.—Inverted P wave in Lead 1; P-R equals .16; normal mechanism; positive P wave in Lead 2; negative P wave in Lead 3; high R in Lead 1; deep S in Lead 3. Possible left ventricular preponderance.

facts as bear upon the subject we are considering. His discomforts had not interfered with his business activities, nor had they given him much concern. At the time of our first consultation his pulse was 100, irregular and alternating. The heart was enlarged, the first sound impure, and the rhythm galloping. Blood-pressure was 180/100. There was a minimal albuminuria, many casts in the urine sediment, and phthalein index 35 per cent. in two hours. The ophthalmoscope revealed

hemorrhagic neuroretinitis, with exudate. After arranging his affairs the patient entered the hospital for observation. A blood-nitrogen estimation revealed urea 28 mgm., total non-protein nitrogen 49 mgm. The blood Wassermann proved negative. After a fortnight in bed on a low-protein salt-free diet his pulse still remained rapid, gallop rhythm and alternating pulse persisted unchanged, and the blood-pressure had increased to 195/120. Mosenthal renal diet test showed inability to concentrate the urine, the specific gravity being fixed at 1.007 to 1.010 chlorid balance minus. The blood count showed marked secondary anemia: erythrocytes, 2,250,000; leukocytes, 14,600; Hb., 53. Kidney phthalein index 24 per cent. The patient is now at the end of the fourth week of hospital observation, and we find his condition as follows: The pulse is persistently rapid, never falling below 100. A blowing systolic
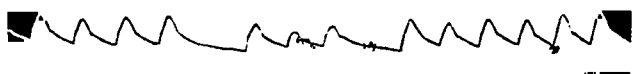
Fig. 3.—Alternating pulse. Premature contractions.

murmur is now present and gallop rhythm, with alternating pulse, persist typically. There is a good deal of soft edema of the feet, gluteal region, and face. The liver is passively enlarged and there is some pulmonary hypostasis. Vision is failing. Urine is abundant, but poor in quality. We find a marked increase in blood-nitrogen concentration, urea 87.5 mgm., total non-protein nitrogen 108.5, blood-creatinin 6.23 mgm. The kidney function index has fallen to 1.94 per cent. in two hours. Blood-pressure 220/130.

CASE III represents the most unfavorable condition in which persistent gallop rhythm is met with. This is a true example of what the French call "rhythmus Brightique." I have followed the clinical course of quite a few cases of chronic nephritis with advancing kidney insufficiency in which gallop rhythm has developed, and without exception they have pursued a steadily unfavorable course toward death from uremia or heart

failure uninfluenced by treatment. Rest, venesection, digitalis, and dietotherapy have alike proved futile, and I have come to look upon the appearance of gallop rhythm with rapid pulse in chronic nephritis with nitrogen retention as marking the advent of the terminal stage. In high blood-pressure, whether it accompany nephritis or be due to causes quite distinct from the kidneys, the appearance of gallop rhythm is to be regarded as introducing a situation of the utmost gravity.

Before closing this brief clinical consideration of gallop rhythm I presume I should for the sake of completeness refer to the so-called protodiastolic gallop rhythm (bruit de rappel, diastolic echo, diastolic double sound) often heard in cases of mitral stenosis. In referring to it I shall merely say that it is an entirely different phenomenon from the presystolic gallop we have been considering, having different clinical associations and causes and entirely different significance. To call it gallop rhythm without qualification is unfortunate and has led to confusion. Clinically, it is met with almost solely in cases of mitral stenosis, although Thayer states that it can be heard at the apex in about 30 per cent. of normal individuals lying upon the left side. So far as I know it is merely of interest as a curious physical sign possessing no prognostic significance. It is heard best in slow-beating hearts and seems to bear no definite relation to cardiac weakness.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

PERICARDITIS WITH EFFUSION

THE patients I wish to present to you today are four in number, all of them illustrative of a disease which is said to be, I think correctly, the most frequently overlooked serious medical disease we have. I refer to pericarditis with effusion. I am fortunate in having a rather large number of cases at my disposal, and these represent different types of the same condition.

Our first patient is a young man, aged twenty, a strong, vigorous individual who has been in the hospital before, suffering with acute articular rheumatism. His principal complaint on entering the hospital, eight days ago, was pain in the left knee, ankle, hip, and right wrist. Along with these he noticed some discomfort about the heart. He describes this discomfort as a "jumping sensation." He is not quite clear in regard to his breathing, but thinks he is a little more short of breath than usual. All of these symptoms date back only a few days. I will not burden you with all the details of the rather elaborate history which we have here, but will say that the symptoms have progressed pretty steadily. In the case of the joints, he is still in the midst of a moderately severe attack of acute articular rheumatism, from which he is suffering quite intensely. During his previous attack of rheumatism he developed a "leaky heart." He does not remember during which particular attack it occurred.

On his entry into the hospital we found the patient somewhat anemic, but well developed and well nourished. Aside from his joint condition, he did not seem to be acutely ill. His temperature has been only slightly elevated, with the pulse-rate

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running from 84 to 110, with no especial abnormalities. Examination of the head and neck was negative, as was also that of the lungs. The heart showed a moderate enlargement with a definite to-and-fro blowing murmur at the apex and a soft diastolic murmur along the left margin of the sternum. In short, he showed the evidences of a moderately enlarged heart with a low-grade double mitral lesion and a low-grade aortic insufficiency. The abdomen showed no muscular rigidity, the
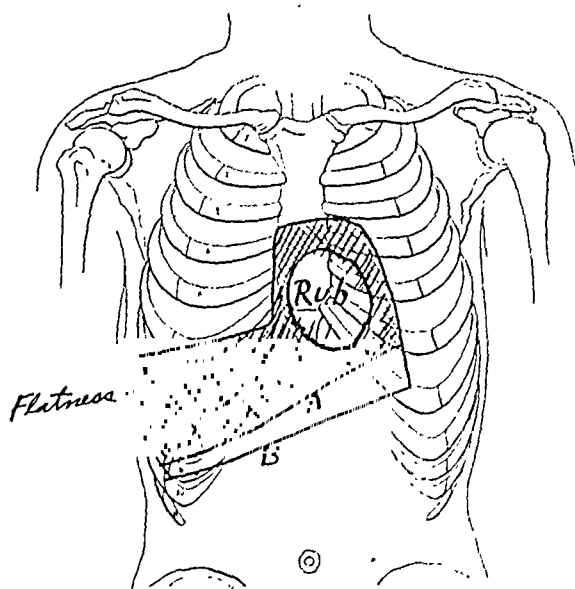
Fig. 4.—Case I.   Diagram showing outline of area of absolute dulness. The line *A* is the position of the lower edge of the liver before the effusion developed; *B* shows the liver edge after the fluid accumulated.

liver and spleen were in normal positions, and there was no tenderness over the liver.   The extremities were negative.

The diagnosis was perfectly simple and read, "Acute polyarthritis rheumatica, with a low-grade double mitral lesion and low-grade aortic insufficiency, in perfect compensation."

On the fourth day after his entry into the hospital a very definite, loud friction-rub was heard over the midsternum. The quality of this and its rhythm did not admit of any doubt

as to its origin. It was a perfectly typical pericardial friction-rub. I examined him on this day and found the outline of the heart absolutely unchanged in every respect. I mapped out the lower border of the liver carefully and found it was not displaced. *There was no increased dulness over the great vessels.* Yesterday, three days later, I dictated the following note:
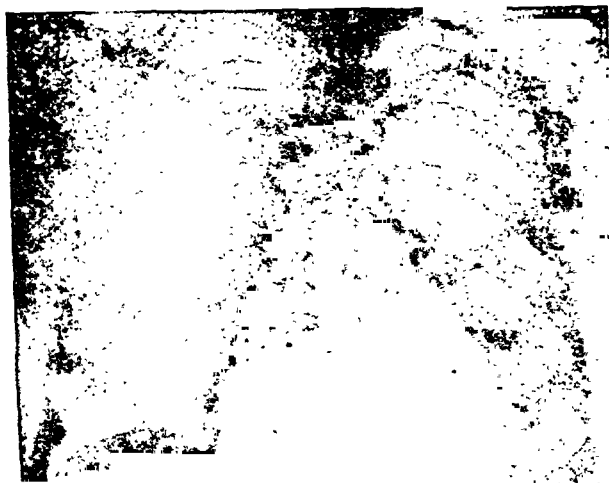
Fig. 5.—Case I. x-Ray of chest, when effusion was beginning.

"Liver dulness is two fingerbreadths lower than it was three days ago. The cardiohepatic angle is distinctly acute; no dulness in the fifth interspace to the right of the sternum; friction-rub very loud and audible over the midsternum and the apex; slight increase of the transverse dulness over the great vessels."

Today the condition is a little bit more marked. The liver is now, as you see, about three fingers below its original position on admission. The cardiohepatic angle is still a right angle or less. There is a moderate dulness over the great vessels at the base and the friction-rub is still audible over the greater portion of the heart. A diagnostic puncture showed a clear serous fluid, so that there was no doubt as to the diagnosis of a pericardial effusion. The x-ray plate, which I am presenting to you, was taken this morning and shows a very typical and characteristic effusion of medium size. When I say that the plate is typical and characteristic, what I really mean is that in these small effusions there is no especially characteristic outline such as I shall show you later in large effusions, but that it is perfectly compatible with a diagnosis of pericardial effusion.

I have chosen this case as a typical illustration of a small pericardial effusion developing right under our very eyes in which we have had an excellent opportunity of watching the entire course of the disease and noting the symptoms and physical signs in the order of their appearance.

As the subject of pericarditis is one in which I have been especially interested for the past five years, you will pardon a little digression to put you abreast of some recent work on this subject. To begin with, the great question in pericarditis has been, *Where does the effusion first develop?* A moment's thought will make it perfectly clear to you that our physical examination will not clear up this matter. If fluid accumulates to the right of the heart, we may perhaps, if we are very skilful, map it out by percussion. The same thing applies to the fluid accumulation over the apex or over the great vessels at the base. Along the lower margin of the heart, however, in the angle between the diaphragm and the anterior chest wall, our physical examination fails to reveal anything abnormal, and the dulness which would be due to fluid there merges into the dulness of the heart above and of the liver below. The same applies to the shadow of the x-ray plate, the dense shadow of the fluid merging imperceptibly into that of the liver below

and of the heart above.   It is this fact that has led to the many
erroneous theories which have prevailed in regard to the place
where fluid first accumulated.   A year or two ago I published
an experimental research on pericarditis with effusion, in which
I showed for the first time that *pericardial effusions accumulate
along the lower margin of the heart, that is, along that portion
of the heart which lies in the angle formed by the diaphragm and
the anterior chest wall.*   Some of you may be familiar with this
work already.   This angle is the commonest location of effusion,
and with small amounts of fluid it is frequently limited to this
area.   As the amount increases it spreads around to the right
margin of the heart and over the apex, and even may be found
filling the prolongation of the pericardial sac which covers the.
great vessels at the base.   In the course of this experimental
research, which was done by injecting cadavers and then mak-
ing models of the injected masses, the striking thing noted was
the frequency with which the left lobe of the liver was pushed
down.

   In the cuts which I am showing you here the three small
pictures in each case represent, A, a model of the heart; B,
a model of the exudate surrounding the heart; and C, a
sagittal section of this exudate.   You can see very prettily
how the fluid accumulates first in the costodiaphragmatic angle,
and then secondly over the great vessels at the base of the heart.
By looking at the middle picture you will see the hole in the
cast of the exudate.   This, of course, is where the heart was in
contact with the chest wall, which is the normal state of affairs.
I wish to lay stress upon this, since the general idea is that as
soon as an effusion develops it pushes the heart away from the
chest wall and causes the pericardial rub to disappear.   Indeed,
I regard this as an error of vast importance, since it leads the
physician to overlook many pericardial exudates of moderate
and small size because he is so firmly imbued with the idea
that a rub cannot exist in the presence of fluid.

   The two figures representing the chest outlines are for the
purpose of showing where the exudate lies with reference to
the heart, so that by means of this diagram and the longitudinal
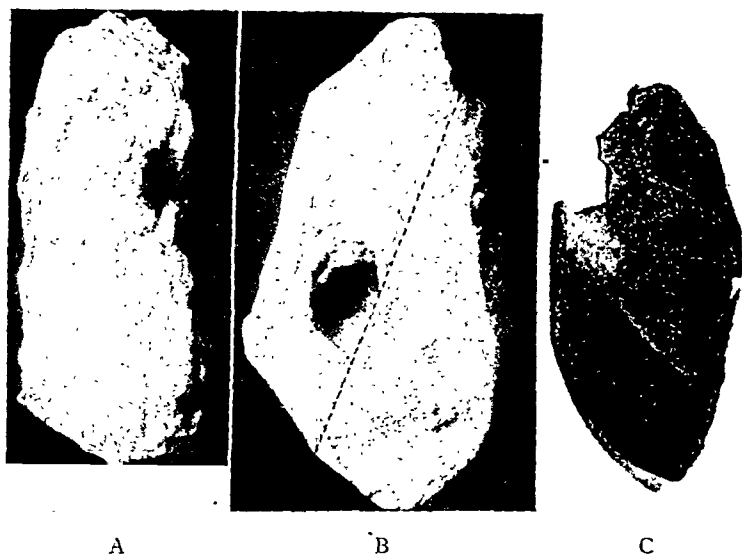
Fig. 6.—Injection 23. Casts of the injection mass (460 c.c. volume). A, Cast of heart; B, cast of exudate; C, sagittal section of exudate. Note the large accumulation in the diaphragmatic angle.
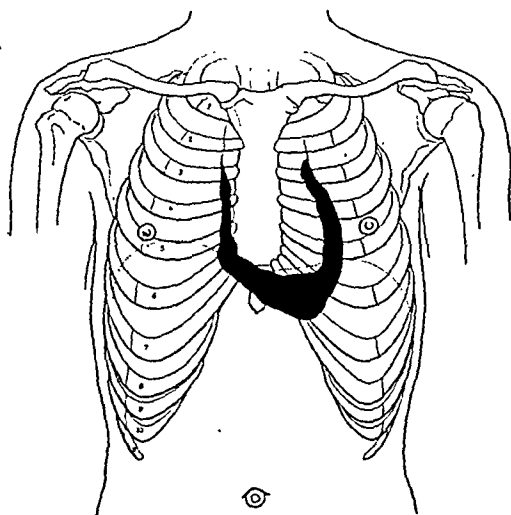
Fig. 7.—Injection 23. Outline of chest, showing position of fluid depressing the diaphragm and liver.
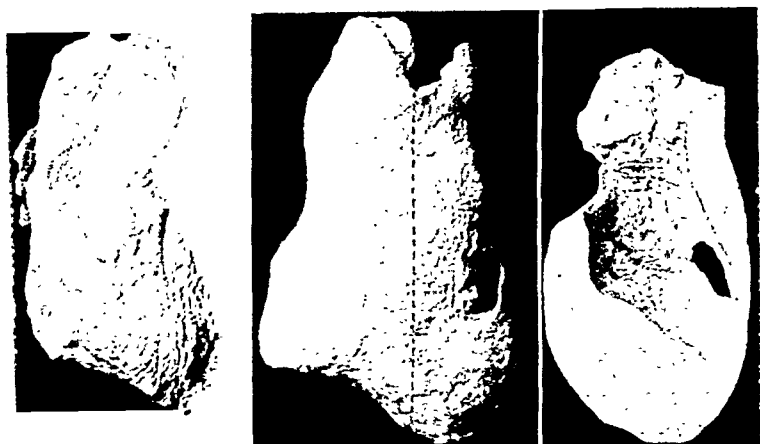
A . B C

Fig. 8.—Injection 24. Casts of the injection mass (575 c.c. volume). A, Cast of heart; B, cast of exudate; C, sagittal section of exudate. Note the manner in which the fluid has accumulated in the costodiaphragmatic angle.
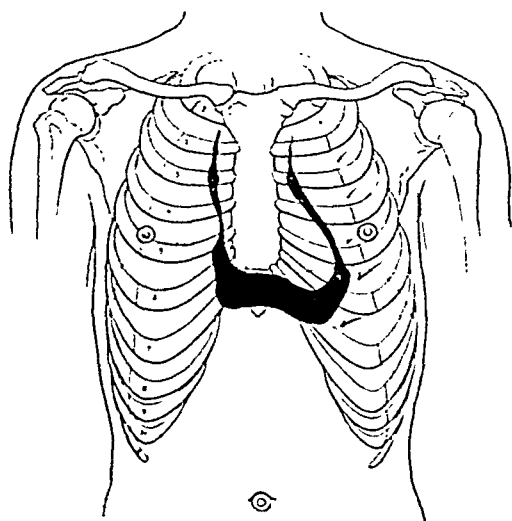
Fig. 9.—Injection 24. Outline of chest, showing position of fluid and depression of diaphragm.

section we can see precisely where the fluid accumulates. A glance at these two plates shows how greatly the liver is depressed even with an exudate of this size, approximately 500 c.c. each. The particular advantage in this method of investigation is its objectivity. The models are made, of course, by mechanical processes and are absolutely accurate. The photographs were all taken at the same time and to the same scale, and the outline of the chest is reduced in exactly the same proportion as the photographs. There is, therefore, no element of subjectivity in this research, as is so often the case where such methods as auscultation or percussion are relied upon.

As soon as it becomes evident where the fluid first accumulates, it is almost a matter of necessity that the diaphragm and with it the left lobe of the liver should be somewhat depressed. Now it has been known since Auenbrugger first discovered percussion that the liver might be pushed down by a pericardial effusion, but it is generally referred to as being somewhat extraordinary and out of the usual. Hale White, writing in French's *Differential Diagnosis*, refers to it as a very unusual phenomenon. Now, as a matter of fact, just the reverse is true. I have watched a large number of cases of pericarditis with particular reference to this point, and not only is it not a rare condition, but it occurs with great regularity, as almost the first symptom of an effusion. The amount of displacement of the liver is not great. A small exudate will often displace the liver but the width of one finger, as determined by percussion; a larger exudate, the width of two or three fingers. This is not anything very striking, and if the liver has not been outlined carefully before, it may easily escape observation. In the case which I have just demonstrated it was the very first sign of fluid which followed the development of a pericardial rub, and was present at a time when the cardiohepatic angle was perfectly normal and there was no increase in the dulness over the base. In other cases which I shall subsequently demonstrate to you the same condition of affairs holds true. It was always the first marked physical sign referable to the effusion.

With this little digression let us pass on to the second case,

which is that of a young Bohemian boy, sixteen years of age, whose case was in many respects very similar to that just shown. He came into the hospital fifteen days ago complaining of pain in various joints, fever, sweats, and chilly sensations; in short, with all the phenomena of a moderately severe case of polyarthritis rheumatica. He was running a temperature of 103° to 104° F., with a pulse-rate averaging 102 to 103, and respirations 26. His previous history was unimportant except that we learn that he had his tonsils removed for repeated attacks of tonsillitis, and his mother states that he had had some heart trouble, which, however, had not prevented him from working hard as an ordinary laborer.

Physical examination on entrance was substantially negative until we came to the heart. We found the apex-beat somewhat diffuse, but plainly displaced about an inch outside the nipple in the sixth intercostal space. The right heart border was approximately normal and the left heart border extended out about ¾ inch to the left of the nipple line. A soft, blowing murmur was heard at the apex and transmitted to the left axilla. The second pulmonic sound was distinctly accentuated. The abdomen showed nothing abnormal; no tenderness anywhere. The liver border was in its normal position. The extremities were negative and the diagnosis made was that of a recurrent polyarthritis with an old endocarditis and mitral regurgitation. The heart was perfectly compensated.

The patient went along in this condition for five days. On the sixth day he suddenly became much worse and developed a distinct dyspnea. His temperature went to nearly 105° F. that evening. I examined the patient the following morning and found the following conditions: The apex-beat was still palpable. The cardiohepatic angle was acute. There was no increase in dulness over the great vessels of the base, but a loud to-and-fro friction-rub could be heard over the larger part of the heart dulness. The liver border was in its normal position and the diagnosis made was acute plastic pericarditis.

Three days later the examination showed a very marked

change, and I noted that the liver was displaced downward three fingerbreadths below its normal position, the cardiohepatic angle being, however, still acute. A very marked increase in dulness over the great vessels at the base was present, and the friction-rub was still audible over practically the entire heart.

Fig. 10.—Case II. *x*-Ray of chest at height of effusion.

This morning an *x*-ray plate was made, which I present to you. You will see that it shows a very large heart shadow with all of the diameters about equally enlarged. It is quite impossible to state just how much of this shadow is due to fluid and how much to the heart, because we had no previous *x*-ray taken, but from our physical examination made on entrance we may be sure that a very considerable portion of this enlargement is due to the heart itself. I feel equally confident, how-

ever, that there is a fair-sized exudate, but would not venture to estimate its size too closely.

Subsequent Course.—He remained in this condition for two or three days, the liver becoming somewhat more depressed, and the dulness over the great vessels of the base becoming more intense. He died rather unexpectedly on the eighteenth day after admission. It is to be noted that the friction-rub
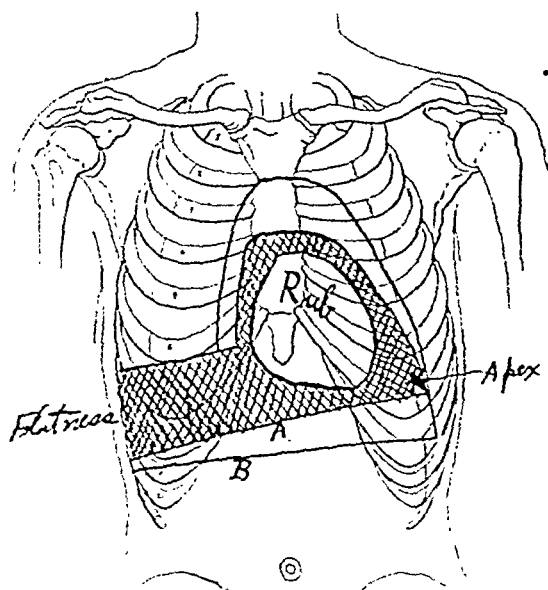
Fig. 11.—Case II. Pericarditis with effusion. A large heart from an old endocarditis. The depression of the left lobe of the liver (*B*) was noted before the rounding of the cardiohepatic angle, or the dulness over the great vessels. The rub could be heard all the time, despite a fair sized effusion.

was heard on the day of his death with about the same intensity as when he first came in. Autopsy showed a serofibrinous pericardial exudate of moderate extent, approximately 300 c.c. in a patient with a large heart due to his old mitral disease.

Let us sum up the salient features of this case: A man came in with a well-defined mitral lesion, with a large heart perfectly compensated and with a recurrent polyarthritis. After some

days he developed a dry pericarditis which rapidly went on to effusion, the effusion looking much greater than it was because of the pre-existing large heart. The first sign indicative of effusion was the pushing down of the left lobe of the liver, and next to this the increase in the previous dulness at the base. Dulness developed in the fifth right interspace somewhat later. The cause for the rather unexpected death may only be surmised, for there was nothing in the autopsy to clearly show what it was. Whether the myocardial change was responsible for the fatal outcome or whether the very moderate amount of fluid together with the large heart produced so much pressure in the pericardial sac as to obliterate the auricles is not possible to say with certainty.

The third case is that of a Bohemian lad, eighteen years of age, a factory worker, who entered the hospital thirteen days ago with the following complaints: He had pain in his chest, great weakness, loss of appetite, and some shortness of breath.

He was perfectly well until three weeks ago, when he began to have some substernal pain and to be very weak. Three days before admission he had a temperature of 101° F., for which he had no explanation. He says that the pain in his chest was substernal and that it was referred to no other point. He said that it was not influenced by coughing and that deep breathing made the pain worse. Dyspnea had been present for some time, but the patient does not know for just how long. He said it caused him no trouble while he was quiet, but as soon as he tried to go upstairs he got out of breath. He noted no tenderness or pain in the abdomen, no swelling of the ankles, no hemoptysis, or other pulmonary symptoms.

His past history stated that he had rheumatism three years previously. He was ill two weeks ago, with the joints very much swollen and painful. He has had a good many attacks of acute tonsillitis, the last one only eight days ago, before his admission to the hospital. The rest of his history is unimportant.

On admission, physical examination showed a young man with a temperature running approximately 101° F., pulse 120, and respirations 26. The blood-pressure was normal. Examina-

tion of the head and neck was negative. The lungs showed no abnormalities. On examination of the heart the apex-beat was found to be somewhat diffuse and difficult to localize accurately. A systolic murmur could be heard following the first sound over the apex, and this was transmitted to the left axilla. The second pulmonic sound was distinctly accentuated. The right border of the heart was just outside the right sternal edge. The left border was an inch farther out than normal. There
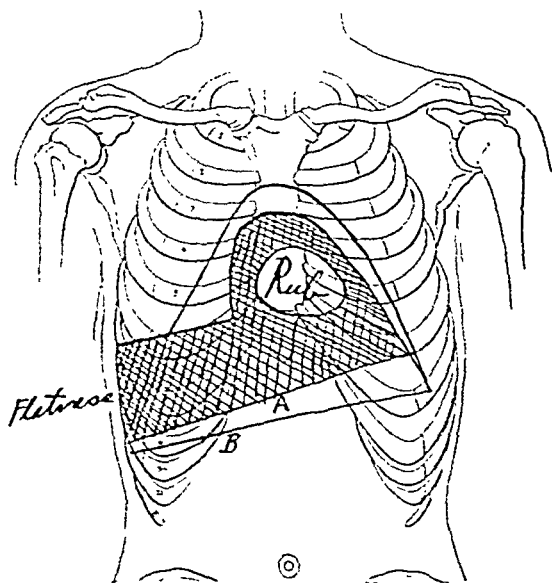
Fig. 12.—Case III. Diagram to show change in the physical findings as the fluid increased in amount. Note the lowered left lobe of the liver and note that a rub could be heard over the area indicated even when the amount of fluid was greatest.

was no increase in the great vessel dulness. Near the left margin of the sternum could be heard a very soft to-and-fro friction-rub, pericardial in origin. The abdomen showed no abnormalities. The liver was not enlarged nor displaced and there was no tenderness. The spleen and kidneys were not palpable.

The diagnosis made on admission was an old mitral lesion

associated with a moderate dilatation and hypertrophy of the heart and a beginning dry pericarditis.

The day after admission the x-ray plate, which I shall show you, was taken. As you will see, it shows a very large area of cardiac shadow, and the question is whether this enormous
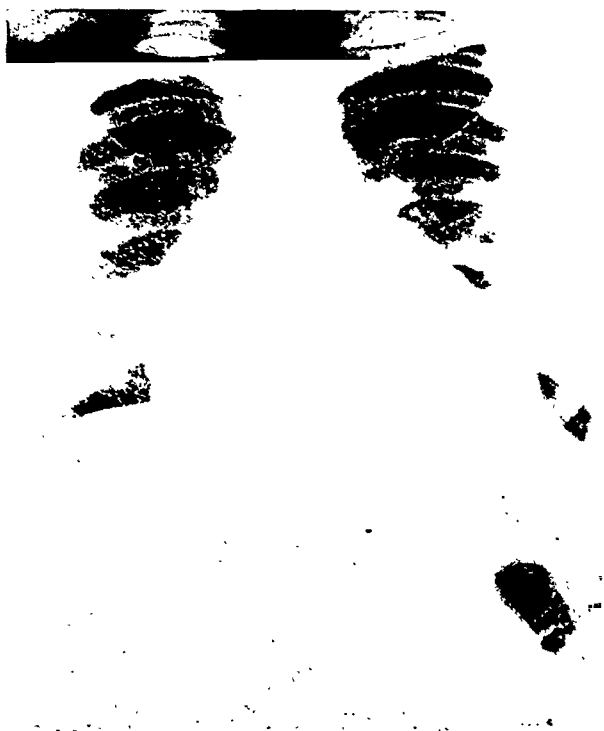
Fig. 13.—Case III. x-Ray taken about thirty-six hours after admission. Probably a considerable amount of fluid present. The edge of liver was a fingerbreadth lower when this plate was taken than on the day before.

heart shadow could be accounted for by enlargement or whether there was not, in reality, fluid at that time. All the diameters were a little larger than on the previous day, and the left lobe of the liver was a fingerbreadth farther down. I am strongly inclined to think that there was actually fluid present in the pericardium from the very beginning.

The subsequent course of the case until today is as follows: The dulness in all directions became more and more marked. The liver became pushed down more and more. The cardio-hepatic angle became markedly obtuse and the dulness extended

Fig. 14.—Case III. x-Ray taken at the height of the effusion. This is the triangular shape, so characteristic of large pericardial exudates. The liver margin was three fingerbreadths lower when this plate was taken than when the first plate was taken.

out beyond the apex-beat, and the dulness over the great vessels became very intense and greatly widened; in short, a typical triangular shadow of a very large pericardial effusion developed. A second x-ray was taken yesterday, and you will see here a very

striking and beautiful illustration of an enormous pericardial effusion.

Today I want you to notice the following points: The liver is pushed down fully four fingerbreadths. The rub can still be heard despite this enormous effusion. If we assume for the sake of argument that a considerable portion of the heart shadow in the first place is due to the enlarged heart, there still remains a very large exudate. The tones are now a little deeper, but not as much as you might expect. Now it is a very typical, rather large effusion, and the only debatable point about it is as to how much fluid was in the pericardial sac on admission.

Subsequent Course.—Two days after the patient was shown in clinic the pericardium was aspirated and 250 c.c. removed. Removal of this fluid made only a slight difference in outline. *The patient apparently improved markedly, but six days later died very suddenly and unexpectedly.* So far as could be told up to the very last minute he was improving. As no autopsy could be obtained, we are a little in doubt as to the exact cause of death. It is a fair question whether it would not have been wiser to have removed more fluid in the first place or to have aspirated a second time. The conditions at the time did not seem to warrant this.

The last patient I wish to present to you is an Austrian machinist, thirty-five years of age, who came into the hospital the day before yesterday. I happened to be in the ward when he was brought up and examined him within an hour of his arrival. He complained of great shortness of breath, pain in the chest and back, and cough, all of which complaints, he stated, had been present only about two weeks. His previous history was absolutely negative, particularly as regards tonsillitis and rheumatism, and the only thing which he ever observed about himself was that he took cold easily. As a matter of fact, he had been advised to leave the city a number of years back because of the ease with which he caught cold, and he had gone to California for this reason. He had never lost any weight and had never had any fever. As already stated, the shortness of breath had been present for about two weeks.

He had pain on deep breathing. The pain of which he complained was situated over the upper portion of the chest on both sides and extended through the shoulder-blades behind. He says it is worse in the morning and is not aggravated by coughing. Occasionally he has a little sputum, which has increased somewhat during the past two weeks. His voice seems somewhat hoarse, but he states that it is no different now from what it has been during the past four or five years. He states that he has never had hemoptysis or night-sweats and has always felt strong and able to work in a garage, doing heavy machinist's work until ten days ago. He says he is heavier now than at any other time of his life.

He shows no abnormal nervous, gastro-intestinal, or genito-urinary sumptoms. His habits have been temperate and he has no syphilitic history. On admission his temperature was 99° F., his pulse 90, and respirations 26. Yesterday the highest temperature recorded was 100° F., and today it has not exceeded 100.1° F. up to 2 o'clock this afternoon. His condition is practically identical with what it was on entrance, so we will go over him together and see what we find.

As you see, he looks to be well nourished and does not look very acutely ill. His head and neck present nothing abnormal. and the mouth shows nothing abnormal with the exception of carious teeth. The tonsils are normal. The chest is symmetric. well developed, and well rounded. The supraclavicular fossæ are somewhat shallow and the clavicles not especially prominent. Examination of the lungs shows that expansion is fair, tactile fremitus is increased somewhat over the right upper lobe. On percussion we obtain a slight relative dulness in both infraclavicular regions and on auscultation we hear moist râles over both upper lobes just below the clavicle. The boundaries all move freely on the left side. On the right side the borders seem fixed and do not move with respiration.

The apex-beat is not to be seen, and with all the care which I can muster I cannot find it by either inspection or palpation. There are no thrills to be felt. On percussion, we find the outlines as I will now put them on the chest for you—absolute

dulness beginning at the upper margin of the third rib. It extends out fully 6 inches to the left of the median line and fully 3 inches to the right of the median line. The cardio-hepatic angle of Ebstein is not rounded. On auscultation, the tones at the apex are somewhat distant, but no murmurs can be heard. Over the base the same thing applies. Along the left margin of the sternum opposite the fourth rib in particular can be heard a well-defined pericardial friction-rub.

The particular point to which I want to call your attention is the outline of the liver. It is exactly where it was when he was admitted the day before yesterday and, as you see on percussion, it is down fully three fingerbreadths below its normal position. The spleen is not palpable. There is no tenderness over the liver. There is no dulness in the flanks. The extremities show no edema. The blood examination showed a leukocyte count of 8900, the differential count of 73 polymorphonuclears, 20 small mononuclear lymphocytes, 6 large mononuclear lymphocytes, and 1 eosinophil. Urinalysis shows nothing abnormal but a trace of albumin.

This case is interesting for two reasons: In the first place it is a beautiful illustration of how a very extensive pericarditis can exist without anybody having very much suspicion that the patient was seriously sick. The condition is so typical as to leave very little room for discussion as to whether the case is one of pericarditis or not. To begin with, he shows exquisitely the depression of the liver, which I believe to be the earliest diagnostic symptom in most instances. Second, he has dulness in the fifth right interspace, although it is not very well marked. We can explain this very readily from the fact that the lungs are firmly fixed on the right side and hence do not retract, so that the Ebstein angle is not rounded as it probably would be in so large an effusion if the lungs were free to be pushed back by the fluid. Across the great vessels we have very intense dulness and the area markedly widened. We cannot locate the apex, but the absence of impulse and the very distinct tones with flatness coming out from the median line make it perfectly clear that we have fluid here, and we are

prepared to diagnose a large pericardial effusion without much more ado. I want you all to come down and hear this loud friction-rub, because again *it shows how very much mistaken the notion is that a friction-rub is incompatible with a large effusion.* Posteriorly over the lungs we found nothing abnormal. This so-called Broadbent's sign, while it often does occur in large effusions, is often absent, and that is the case here. I do not regard it as ever occurring except with quite large effusions, and
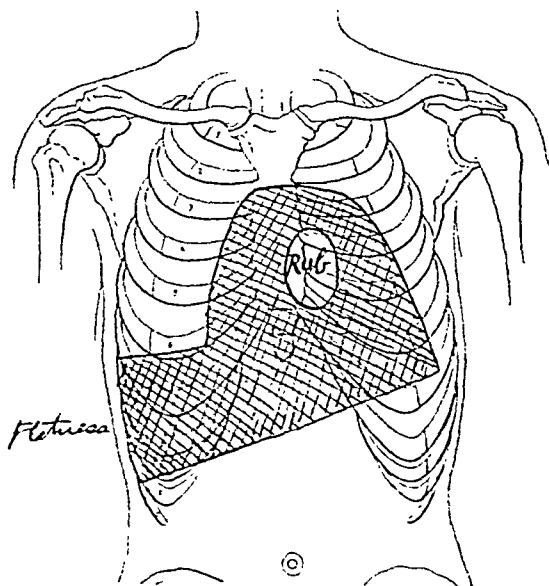
Fig. 15.—Case IV. Tubercular pericarditis with large effusion. Note the downward displacement of left lobe of the liver. Despite the large exudate, a rub is still audible.

then not regularly. The nature of this effusion can hardly admit of much discussion.

We have here all the findings of an old as well as of a recent tuberculosis of the lungs. The adherence of the right lung shows an old pleuritic process and the numerous râles in the infraclavicular region show recent changes, so that we are not surprised to find that in the small specimen of sputum we had no difficulty in finding tubercle bacilli. The fact that the

pericarditis is of a tuberculous nature is also borne out by the white count not being increased above normal. As I show you the very excellent x-ray of his chest it is hard to believe that so large an accumulation of fluid could occur with comparatively slight symptoms. The reason for this is to be found in the gradual accumulation of fluid. The pericardium is, in reality,

Fig. 16.—Case IV. Tubercular pericarditis. Large effusion. Compare the great size of the heart with the paucity of symptoms.

a pretty tough membrane. This is additionally proved by my injection experiments, when I tell you that I injected over 100 cadavers and in two-thirds of them I actually ruptured or stripped up the pericardium endeavoring to get in larger quantities of fluid than it will hold. In the pathologic processes of the pericardium which develop slowly the membrane becomes

injected and softened and is capable of very great distention without producing high intrapericardial pressure. Were this same sized exudate to be poured out in a short length of time, the probabilities are that the circulation would come to a standstill very promptly owing to the compression of the auricles.

Now in the light of these 4 cases let us compile a little synopsis of the newer aspects of the disease.

First, In pericarditis with effusion where the amount of fluid is sufficient to be recognized clinically (this generally means 150 to 200 c.c.) the left lobe of the liver is in the great majority of cases the earliest diagnostic sign of effusion. If there is no cardiac break-down to produce an enlarged liver, the combination of pericardial rub with the pushing down of the liver one or two fingerbreadths, is in the highest degree diagnostic of effusion. The more carefully you watch for this sign, the more frequently you will find it.

Second, The depression of the liver antedates in the great majority of cases the development of the dulness in the fifth right interspace (Rotch's sign) or the rounding of the cardiohepatic angle (Ebstein's sign).

Third, It usually antedates any increase in the dulness over the great vessels.

Fourth, The most valuable feature of this new sign is the ease and accuracy with which it can be determined. As is well known, the outlining of the heart cannot always be done with great accuracy, but light percussion will determine the lower margin of the liver in the median line with great precision and ease.

Fifth, From the standpoint of finding fluid with certainty, a puncture just to one side of the ensiform cartilage (Marfan) is most certain to find it. This point, however, is of disadvantage when done for diagnostic purposes, for in the event that fluid is not found, the right heart is likely to be punctured in the most vulnerable spot. I prefer to go in just outside the apex, directing the needle upward, inward, and backward toward the hilum of the lung. In the event the heart is punctured in this position the left ventricle is the part met with, and a puncture here does very little damage. As a matter of fact, the danger of wounding the heart is one which is greatly overestimated.

CLINIC OF DR. RALPH C. HAMILL

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COCCYGODYNIA

OPPENHEIM[1] thus describes coccygodynia: "The name coccygodynia is given to a severe neuralgiform pain in the region of the coccyx which occurs almost exclusively in women. The pain comes on spontaneously or in the act of sitting down, of walking, or of emptying the bladder and intestine, or it is increased by any factor which is associated with contraction of the muscles which are inserted at the coccyx. The coccyx is usually sensitive to pressure and to movement. The disease follows a severe confinement or trauma, but it may have an insidious onset. It has even been observed in children. It may develop without any exciting cause in hysteric persons. In many cases it is a true neuralgia; in others it is due to an inflammatory process in the muscles inserted at the coccyx, or in the surrounding soft parts or bones. These conditions can usually be distinguished from neuralgia by careful bimanual examination. I have seen slight cases recover in a few days or weeks, *c. g.*, under the influence of opium suppositories, others in which the refrigerating double current sound was helpful, and severe cases which defied all treatment and necessitated an operation (separation of all the soft parts from the coccyx or its removal)."

One wonders just what is meant by a "true neuralgia," and just what the author means when he says "it may develop without any exciting cause in hysteric persons."

The case I wish to tell you about today is of interest because it shows that what might be considered a real physical cause

[1] Bruce's Translation of the 5th Edition of Oppenheim's Text-book of Nervous Diseases, p. 595.

may not, in the ordinary sense of the word, be one at all, and just as the origin was not traumatic, physical, the trauma of surgical interference was not curative. It shows the futility of surgical procedure and various other factors of interest when we consider the patient as an individual and not as a mixture of organs and tissues.

The patient is a young woman twenty-eight years of age. She was brought up carefully, though perhaps without adequate instruction in sexual matters. Though rather delicate as a young girl, she has now developed into an unusually strong, healthy woman. For several years past she has worked, closely associated with men. She is unmarried. There is nothing in her family history of special importance. When she was about fifteen she fell and was very badly frightened, though not severely injured. Two or three weeks after the fall, which was not followed by any local discomfort, she woke in the night with a severe pain in the region of the coccyx. Since that time the pain has been more or less disabling: at times she would be overwhelmed by it, at others practically free for a month or two. Of late it has bothered her so much that she has begun to fear ideas of suicide. She has always been partially or completely relieved by an enema, but, naturally, it was frequently not at all convenient if the attack should come while at work or on the train or at a party. She thought the pain was brought on by hard feces in the rectum, gas, "congestion of the bowels" (that certainly sounds as though some physician had spoken). She could not sit on folding chairs, as she said, "in church or Sunday school." She was unable to lean back and sit at rest, to allow herself to day-dream; and any strong emotion, worry, anger, or any other intense feeling might cause the pain.

The pain might be momentary or it might last two hours. She thought it might have something to do with "poor vitality." There is a tender spot in the perineum, pressure about which might start the pain. The coccyx has been removed, but she has had the pains just as before. For six months before I saw her she had had considerable dysmenorrhea.

This, in effect, is the history. The emphasis was put on

the accident and injury at fifteen; on the causation of the pains by pressure of hard objects, uncomfortable chairs, feces; the spot, touching of which caused pain; the relief of pain with enemata. Poor vitality was twice spoken of, both as a cause and as a result of the pain.

But here was an unusually healthy appearing woman, too healthy to be thought of as suffering for the past thirteen years from anything organically serious. Therefore one must inquire as to the actual value of the various items of her history and try to fill in some of the blanks. First, there is the trauma. She thought she might have bumped her coccyx, but she did not remember any local discomfort as an immediate consequence. Therefore are we not perhaps privileged to deprecate its importance? If there had been a fracture she would have had very severe discomfort within twelve hours after the accident. If there had been a bruising, a periosteal or a subcutaneous hemorrhage, the same is true. There was no trouble of this kind. There was, however, a severe fright at the time of the accident, there was a psychic trauma, and when we have heard the finish of this story and realized the rôle played by the mind, especially in a state comparable to fright or panic, we may perhaps conclude that this psychic trauma is of more importance than the physical.

The pain came on two or three weeks after the accident. The patient is a bit hazy as to this first pain—whether it woke her out of her sleep, whether she was just going to sleep, she does not seem sure. If it did come on during this period of dulled consciousness it will fit in with our ideas as to the beginning of these symptoms which may be classed as related to bad habits, tics, dysmenorrhea, etc. At any rate, the fact that the pain did not begin for two or three weeks after the trauma certainly weakens the importance of the accident from the physical standpoint.

Now the description as to when the pain would come on is the usual one, the one to be expected from anyone who has had to tell this story to doctors who are content with the statement of the patient. One wonders, however, why she does

not have the pain every time she sits, especially on an unupholstered seat; also, if the presence of feces and gas in the rectum cause a pain of such disabling power, why they do not cause some kind of discomfort whenever present. In other words, there are inconsistencies that should discount the story and demand further light.

How are we going to get the light? That is a matter to be approached differently in each case. The first item that led me in the right direction was a statement that seemed rather to slip out in a moment of relaxed caution, namely, that the pain was like a cramp.

If the pain was like a cramp, what was there about it to justify the panic into which it seemed to throw her? She said that if the pain were to come on where she could not do something right away she did not know what to do. In other words, the statement and the manner of making it combined to give a definite conception of the panicky state of mind induced by the pain.

Now if one thinks for a moment of the difference in sensation in the skin around the genitals, perineum, and anus from that in the skin of the abdomen, chest, or arms, one realizes that it may be due to the fact that sensations coming from this region are endowed with a different cerebral element. If this is true of the skin may it not also be true of the underlying muscles? May not movement of the perineal muscles be sensed differently from movement in the legs and arms? May movement of muscles attached to the coccyx or to the fibrous central point of the perineum be felt in a different way from movements of muscles—say, about the shoulder-joint? What is it that gives the feeling tone to the sexual orgasm if it is not some form of muscular spasm? There is a condition called vaginismus by which is meant a spasm of muscles about the vagina when intercourse is attempted. It is ordinarily considered as an effect of frigidity. In some cases I believe it to be an abortive orgasm. It is the spasm of muscles which when rightly timed and conditioned makes up the orgasm and amounts to a voluptuous sensation. When it is premature and improperly

conditioned it is described as something to avoid—a pain; something to be ashamed of—a weakness. Is the pain described here of this order? How can we obtain an answer to this question without giving offense? The patient was asked to tell all the occasions she could remember when she had had the pain.

After two or three visits, at which times the list was added to, it was evident that as a usual thing there was a complete absence of physical cause—hard chairs, etc.—and there was present an element of emotional value accounting, possibly, for the occurrence of the pain. For example, on one occasion the pain had come when at a wedding of a very dear friend; on another when a man physically and mentally attractive to her (her words) stood beside her looking over her shoulder; another when reading an exciting love story just before the man and woman were to fall into each other's arms; another when she dreamed of a certain man with whom she was on close business terms. Occasions in which an element of this sort occurred were more frequent than those in which there was no possible sexual stimulant suggested, so that when instances of the pain were asked for, statements such as of hard chairs, leaning back, hard feces, and so on, were not mentioned. Are these factors perhaps merely the patient's reflection of the ideas of the various physicians and surgeons she had consulted?

A second item that deserved attention was that the pain seemed so closely associated with the idea of panic. Why is that? Does a person get in a panic if he has a cramp in his foot? I have never met one. There is something about this pain that causes panic. May it not be because of its location? It cannot be seen, for one thing. For another, and probably much more important, it comes from that part of the body that has been made as strictly taboo as possible ever since earliest infancy.

Just what is meant by this? The meaning is that the perineum, the anus, and the genitals are parts of the body almost never referred to by any human over five or six years of age. The actions of children of three, four, or five show a curious

combination of immodest smartness in exposure and fearfulness of detection therein, and no one seems to have the ability to cast their mind back with sufficient clarity to get to the origin of these ideas of shamefulness. As I have watched the infant on its mother's or nurse's knees, having its diapers changed, it has seemed to me that perhaps here I saw one of the original sources of the matter. As the diaper is removed the infant's hands are very apt to go to the genitals, and when they do there is almost certain to be an expression of disapproval on the mother's or nurse's face. Very frequently the intentness of the infant's inspection of the overhanging face is most striking. At that age words mean nothing; expression means everything. We all know how readily an infant will howl at a frowning, and chuckle at a smiling, face. In short, I believe before the age of eighteen months a very strong sense of the improprieties has been established; a background that makes valid the later corrections, a sense that weighs, appreciates, and gives force to all references, suggestions, and admonitions relating to this region. It is easy enough to understand the development of ideas when they come in response to words, to speech, because that is the current coin in which we as adults most consciously deal. Our thoughts are put into sensory images, into words, and because they are sensory images, things we seem to see and hear, they seem much more real, much more important than feelings, prejudices, and attitudes of mind. A prejudice relating to this region of the body finds something similar in every human it comes in contact with, finds justification on all sides; hence, it has a force, a power compared to which words, frank expressions, are rather futile.

It is in something similar to the foregoing that we are to find the explanation for the panic element involved in, or induced by, the sensation described as pain by this patient.

Another item in the history is of interest as bearing on this attitude of mind, namely, her statement that the pain had something to do with "poor vitality." This was her phrase. The pain was brought on by poor vitality or, what she emphasized as the more important, was followed by poor vitality. There

is something reminiscent about such a statement. It sounds like the advertisements of the patent medicine men and the quacks—"poor vitality brought on by masturbation."

When I had assured myself as to the relationship between the panic, the pain, and the sexual or moral ideas of the patient, I showed her anatomic charts of the perineum and the genitals and described the function of the muscles. This necessitated going into the function of the bulbocavernosus and its relation to erection and copulation. This information was received with interest and without offense.

Then the fact that the first pain occurred at fifteen was discussed, and the question was raised as to what girls of that age might be thinking about. Among other things masturbation, either manual or through voluntary setting of the muscles about the perineum and thighs, was spoken of, though merely as one of a number of things.

Several visits intervened, and then the patient remembered that though she could not be sure about the first pain she did remember, that a few weeks thereafter she had tried to get a pleasant sexual sensation in some manner. She could not remember particularly as to whether she had gotten it or not, but she did remember having this pain and being driven into a panic thereby.

Now there seemed to be enough evidence to justify the conclusion that the pain, the panic, and the despondency were all on a more understandable basis, and it seemed reasonable to say that the pain was the cramp-like contraction of muscles in the perineum, those that are concerned in the essentials of copulation, namely, erection and orgasm. This was pointed out to the patient and linked up with that which has been said concerning the mental and moral attitude toward the whole realm of sex and the sensuous. Finally, she drew the conclusion that perhaps the pain was, in reality, part of the *normal* sensation of sexual excitement.

After the second or third visit I think the patient felt that an attempt to understand and really remedy the condition was under way. At any rate, she ceased to have the pain with

anything like the same intensity. The last three times she had the pain she immediately got hold of herself, told herself there was nothing to get panicky about, and the pain passed off. It is my belief that this improvement will continue.

The most satisfactory thing about the case is the general emotional change in the patient. The despondency has passed into a state of buoyancy and she declares there is a load off her spirits.

In conclusion, I would say that this case of so-called coccygodynia is really a sort of vaginismus which, in turn, is closely related to the muscular condition of erection and orgasm. The muscular conditions have their emotional tone fixed by the location of the sensation—they are in the perineum, they are normally voluptuous and pleasant, but in a condition such as exists in this case they are painful and induce panic.

Surgical procedures may be of benefit, but I strongly suspect that all cases of coccygodynia are of much the same nature and can be far better handled with understanding than with anesthesia and blood-letting. In that respect I would emphasize the change in emotional status especially. Contentment has taken the place of despondency, and I do not believe that would occur as the result of a surgical procedure, no matter how successful.

To return to Oppenheim's definition, I would be inclined to add that coccygodynia is closely related to vaginismus, is increased by contractions of the muscles inserted into the central fibrous point of the perineum. Also, as regards the statement "these conditions (local inflammatory processes) can usually be distinguished from neuralgia by careful bimanual examination." I would say that in these cases there may be such wincing from any examination that the surgeon might easily be misled into thinking that there was inflammation, when, in reality, the real trouble is in the mental attitude.

ADHESIONS INVOLVING THE CAUDA EQUINA

This is a case of disease of the cauda equina. The diagnosis was made, the patient operated, and the morbid process found. The character and distribution of the lesion made it impossible to accomplish complete removal, and so the therapeutic result aimed at was not achieved. This man is forty-five years old. He is single. The family history is of no importance medically. There are no factors outside this history that seem to bear upon his trouble.

In 1907 he thinks that he had tuberculosis. For three years he was in the South, during which time an abscess appeared and broke just above the inner third of the right clavicle. This discharged for some time, but finally healed. The next year the glands in the right axilla became inflamed, apparently broke down, and an operation was necessary. This was in 1910.

That same year he went to the Pacific coast. Shortly after his arrival he began to have what was called "sciatic rheumatism." This name was given to a pain that radiated down the backs of the legs into the calves. At first this pain was intermittent, but after a few weeks it became practically constant and required morphin. Because of this pain he went to Nevada, thinking that it had something to do with tuberculosis and that the climate in Nevada was the most salubrious. While in Nevada he thinks that the tuberculosis healed, but in spite of a general improvement in his condition, a weakness suddenly developed in the right knee. This was about a month after the beginning of the pain. Two weeks later the left knee weakened. The weakness and pain in the legs and in the buttocks, though interfering greatly with his getting about, still were not entirely disabling, but one morning a few weeks after both knees had become involved he awoke paralyzed from the waist down. He was unable to feel the bedclothes or other pressures on the legs; the paralysis was of sensation as well as motion. After a few months a masseur began to

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work on his legs, and with two or three weeks' work the left leg began to come back, and then the right.

A wasting away of the lower legs was observed shortly after the complete paralysis appeared. At no time was there sufficient trouble with micturition to impress his memory.

The atrophy went on rather rapidly in the right leg, involving also the thigh and the buttocks; it was only of the muscles below the knee in the left leg.

For the past ten years the condition has been practically stationary. There has been great motor weakness in the whole right leg and in movements of the left lower leg. However, he has been able to get about with a cane.

The principal symptom during these past ten years, that which led him to consult me, was attacks of very severe pains. These attacks came every four or five weeks, they lasted from twenty-four to thirty-six hours, each pain lasting two or three minutes. The pains run down the right leg, mostly along the inner surface, and are most severe in the foot. They are described as "like sticking a knife into a nerve center."

Examination.—The patient walked into my office with the aid of a cane. His complexion was rather sallow; otherwise he did not seem to be suffering from any acute or progressive disease.

During the office visits, which totaled about two and a half hours, he had no pains and said that for the past few weeks they had been less severe in character.

The cranial nerves were normal. Motion and sensation were normal in the arms. There were the scars of the abscesses cited in the history above the right clavicle and in the right axilla.

The gait showed a peculiar mixture of foot-drop and of failure of the muscles of the calf. It was somewhat as though he walked on peg-legs.

There was a very marked wasting below the knees on both sides, and when he turned his back the picture was very striking and unusual. The right buttock and hamstrings were greatly wasted, the adductors were fairly well preserved, so

that the limb had almost a normal profile, but seemed greatly lacking in fulness.

I failed to obtain a response to the faradic current in either leg. Also, there was no response to the galvanic in the right and in the muscles below the quadriceps femoris in the left. The last named muscle responded rather weakly to the galvanic current. It is to be remarked that the patient was quite tired when he came to my office, and this may have accounted in part for the marked change in electric reactions. At the time of this examination the left knee-jerk was not to be elicited; during his stay in the hospital, when he was well rested, the reflex was demonstrated.

To particularize a little, there was marked atrophy of the peroneal and calf muscle groups in both legs, also of the right hamstrings and gluteals. There was a moderate degree of atrophy of the right quadriceps femoris.

There was very slight active movement in any of the muscles of the lower legs. Extension and flexion of the right knee and extension of the thigh were weak; adduction was fairly well preserved. The left thigh muscles were not involved.

Passive movements of the ankles were but little interfered with; of the other joints not at all.

The ankle- and knee-jerks were abolished. The abdominals and cremasterics were normal. There was no Beevor sign. Motion and reflex were normal in the arms.

Sensation of all varieties was lost in the right lower leg: there was some appreciation of pressure on the posterior surface of the right thigh, but lighter touches, pain, and temperature were lost over the sacral distribution up to the line that runs from about the sacro-iliac synchondrosis to the outer end of the gluteal crease, and following in that same general direction to just above the outer surface of the right knee—the line of the first, second, and third lumbar segments.

There were no signs of cyanosis or of other trophic disturbance except the atrophy and an enlarged nail on the right great toe.

The essential symptoms, then, were the atrophic paralyses

of the muscles of the left calf and tibial groups, of all the muscles of the right leg up to and excepting the quadriceps femoris and adductors, the sensory loss, and the pains. The symptoms conspicuous by their absence were those referable to the bladder function, vasomotor paralysis, scars of pressure sores, and loss of sensation in the left leg.

Diagnosis.—Usually in diagnosis of disease of the central nervous system we want first to determine the location of the disease. In some conditions we are aided in diagnosing the nature of the lesion by its location; in this case, however, it seems as though we might first determine the nature of the disease and that this will help somewhat in placing it.

Pain felt in the area of distribution of a nerve root, followed by paralysis of the parts below this region, is due to a focal lesion, either a localized inflammatory process or the pressure of a tumor. Which have we to deal with here?

From the history of the abscesses in the neck and axilla followed directly by the acute period of the present condition we need not hesitate to presuppose an inflammatory process. The predilection of tuberculosis for the spine is well known. Hence, when we reach the stage in this history where the patient complained of "sciatic rheumatism" we must, perforce, think of a tubercular process of the spine.

If now we settle on the underlying process as one of inflammation, probably tuberculosis, where are we to localize it?

Obviously, it is below the level of the third lumbar segment of the cord. Is it of the cord or of the roots thereof?

In the first place, the initial pain was in both legs, hence the inflammatory process was of sufficient extent to involve the posterior roots on both sides of the cord. Such an extensive inflammatory process, later producing paralysis, would cause a very serious destruction indeed if it involved the spinal cord. Inflammatory involvement of the cord would probably cause a transverse myelitis, a very severe paralysis, marked trophic changes, and, above all, distressing bladder disturbances.

The actual paralysis is bilateral. Hence, if we postulate an involvement of the cord itself we would certainly expect

marked trophic and bladder disturbances. If the cord involvement was of the conus and the inflammatory process extended to the laterally lying roots, that is, the second, third, and fourth lumbar, a picture might be produced presenting the motor and sensory symptoms of this case, but the picture would be decidedly changed by the addition of bladder symptoms—involuntary or inhibited micturition; also, it is probable that the quadriceps femoris would be more seriously involved than it now is.

In view of the history a diagnosis of inflammatory involvement of the intradural structures was made. Because of the muscles involved and of the level of the sensory disturbance this lesion was believed to include either the cord up to the third lumbar segment or the roots coming from the lower lumbar and sacral portion of the cord. The character of the pain meant that there was an involvement of the lower lumbar or upper sacral *roots*. The radicular, or root, pain is characteristic. It is momentary or lasts a minute or two; it is described as a burning jab, it recurs frequently during attacks lasting a day or two and coming every month or two. It recurs frequently in the same place, and in this area of pain there is a diminution of sensation.

When the lower end of the cord is involved, bladder disturbances are the rule. They are absent in this case. Their absence is the strongest item upon which the differential diagnosis between disease of the cord and disease of the cauda is made.

A diagnosis of adhesions involving the cauda equina at the level of the fourth lumbar vertebra was therefore made.

Description of Chart I.—This chart shows the relations of the lower end of the spinal cord and the lumbar and sacral roots to the vertebræ.

The cord ends at about the upper border of the second lumbar vertebral body. The roots forming the cauda equina lie on the posterior surface of the vertebral bodies until they reach their exits in the intervertebral foramina at the lower face of the corresponding body.

As you can readily see, if a lesion originating in. or from

around, the fourth lumbar vertebra were to so spread as to involve the roots of the cauda, exactly the picture would be produced that our case shows. The knee-jerks are both affected, the right lost, the left much diminished or lost. The quadriceps femoris and the adductors are somewhat weakened perhaps, but do not share in the devastation exhibited by the muscles innervated by roots below the fourth lumbar. In other words, the second and third lumbar roots, escaping, carry sufficient trophic fibers to preserve these two muscle groups.
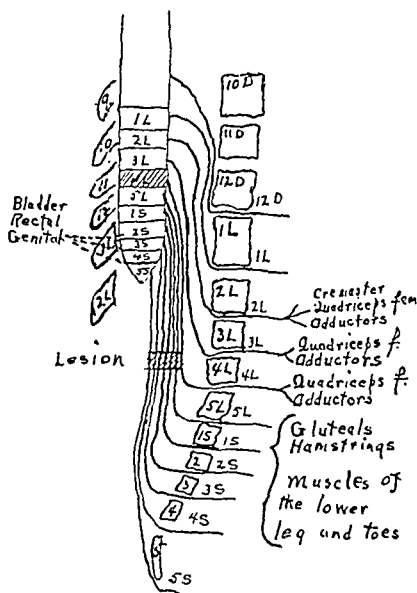
Fig. 17.—Chart I.   (See text.)

In the left leg the gluteals and hamstrings are not nearly so badly damaged. In fact, it is difficult to say that they are really involved. In the right, however, the gluteals, hamstrings, and muscles of the lower leg and toes are badly atrophied—all muscles whose nerve or trophic supply is from below the fourth lumbar.

Description of Chart II.—If now we look at the chart of the sensory disturbance we find that it follows very closely the

lines of the first, second, and third lumbar segment.   Sensation is normal down to this line.

The affected muscles are supplied by the 5 L., 1 S., 2 S., 3 S., and 4 S. segments.   The muscles are atrophic—there is a destructive process in the central nervous system as the cause. We have already decided that the causative lesion was an inflam-

Very marked loss of forms of sensation in right lower leg: areas innervated from L4, L5, S1, and to less extent S2. Partial loss in area of S3. Unable to demonstrate loss in S4 and S5.
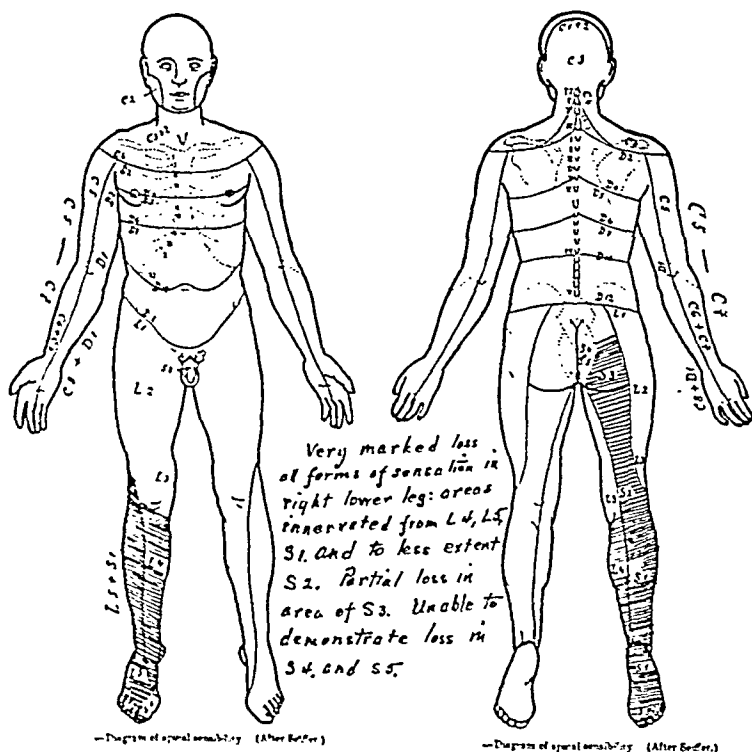
Fig. 18.—Chart II.   (See text.)

mation. If we suppose this process to involve the cord, where I have shaded the fourth lumbar segment, instead of the cauda there is no reason to suppose that an inflammatory lesion would not have involved some of the laterally lying roots. If the lesion were of the fourth lumbar segment of the cord, then we would certainly expect a more irregular picture of motor and sensory disturbance. The upper line of their disturbance

would not be the same. There would certainly be more involvement of either the anterior or the posterior roots.

In fact, if we postulate the lesion as inflammatory, it is difficult to imagine it at any level other than at the level of the fourth lumbar vertebra.

Prognosis.—Because of the non-progressive character of the disability the patient was told that very little could be hoped for from operation, and that the only reason for operating was to relieve the pain. If the pains were too severe, too unbearable, then it was reasonable to seek relief in operation. However, since it seemed probable that tuberculosis was the underlying cause, the patient was told that if an operation was decided upon it should be done only under local or spinal anesthesia; that he should not be exposed to a general anesthetic lasting for an hour and a half to two hours. It may be that this precaution was unnecessary. At any rate, through some mischance he entered the hospital and was operated without my knowing of it beforehand, ether anesthesia being used. The only reason I speak of it now is because I believe the idea is a correct one.

Operation.—Here are the surgical findings as recorded at the time of operation: There is a tumor mass, at the level of the fourth lumbar vertebra, about $1\frac{1}{2}$ inches in length, occupying the entire lumen of the dural canal, and consisting of nerves and fibrous tissue bound together in an almost inseparable mass. The enveloping dura is firmly adherent to the mass. Above this mass the cauda and cord are free. There is considerable cerebrospinal fluid under increased pressure. Below the mass the subdural space is clear.

Some effort was made at separating the different nerves free from the mass. However, it did not seem advisable to go on, and the operation wound was closed.

For the first few days after the operation there was a loss of bladder and rectal control, due, no doubt, to the manipulation of the sacral roots in the effort to free them from the adhesions. Control was regained after about a week, but the persistence of pains did not lead one to expect great therapeutic benefit.

ESOPHAGEAL DIVERTICULUM

THE cases that I am to present to you are from my private service at St. Luke's Hospital.

Case I.—The first patient, referred by Dr. Rutledge, Mrs. H., aged forty-five, married, entered the hospital on March 29, 1921, complaining of pain and tenderness in the left hypochondrium. She also complained of nausea and vomiting, constipation, prostration, and weakness. She has frequent distention of the abdomen with belching of gas.

Onset and Course.—On March 29, 1920 the patient had a sudden severe pain in the left hypochondrium which lasted until it was relieved by morphin. After the attack the patient became very weak and nauseated, and was finally relieved by vomiting. The vomitus was dark brownish in color. In the last year there have been seven or eight of these attacks, and these have increased in severity in the last few months. There was no associated chill nor was there any fever. During the attacks the abdomen became markedly distended and was partially relieved by belching. Following all of these attacks there was marked prostration.

Pain.—The pain is felt to the left of the midline below the costal margin, but at times it becomes diffuse over the whole abdomen, and then seems to cause great pressure in the cardiac region. There is tenderness just to the left of the midline below the costal margin.

Nausea and *vomiting* seem to be only present during the attacks of pain, and on two occasions a dark brownish vomitus was produced. The vomiting seems to relieve the pain. The last attack of vomiting was four weeks ago.

Belching.—In the last four years the patient has had some distention of the abdomen, usually coming on at midnight and lasting for two hours. Of late this has occurred two or three hours after eating. The feeling of distention is relieved by soda and by belching.

Diet.—Strict diet of various kinds has not relieved this trouble.

After the attacks the patient has been markedly prostrated, so that she has been confined to her bed for several days at a time. There is a tendency to constipation. Between the attacks the patient feels very well. Her appetite is good and she is free of symptoms.

Past History.—She has had the usual diseases of childhood; malaria several years ago. Twenty-eight years ago she was supposed to have had lung trouble. During that time she had fever for a year and a half. Seventeen years ago her appendix was removed, and this was complicated by phlebitis on the right side, which was followed by varicosities. She has worn a rubber bandage ever since.

Family History.—Her father died at the age of seventy-three of diabetes; mother died at sixty-four of organic heart disease. Three sisters are living and well. One sister died of brain abscess. One brother is living and well. There is no history of tuberculosis or carcinoma in the family.

Marital History.—Her husband is living and well. She has one daughter living and well. There have been no miscarriages.

Menstrual History.—She began to menstruate at fourteen years, twenty-eight-day type, lasting three days. During the last year menses have been somewhat irregular.

Physical examination reveals a rather well-nourished white female of forty-five years, who is not acutely ill. She is able to be up and about without difficulty. There is no sign of eye disease. The nose is negative. Teeth are in good condition, including examination by *x*-ray. The tonsils are absent and there are no adenopathies. The thyroid gland is not enlarged. There are no signs of Graves' disease. The lungs are negative.

The heart is slightly enlarged; there are no murmurs and the pulse shows an occasional irregularity. Blood-pressure measures 112 systolic and 80 diastolic. There are no Virchow glands to be found in the neck. There is no enlargement of the superficial lymphatics.

Abdomen.—There is a large amount of adipose tissue. The muscle tone is poor. There is some tenderness on the left side below the costal margin and extending downward along the colon. No mass can be felt. The liver is not enlarged and the spleen cannot be felt.

Reflexes are sluggish, but there are no abnormalities.

Laboratory Findings.—Red blood-cells, 4,650,000; white blood-cells, 12,100; hemoglobin, 92 per cent. Differential white count shows small lymphocytes, 25; large lymphocytes, 2; polymorphonuclear neutrophils, 72; polymorphonuclear eosinophils, 1.

Urine shows a faint trace of albumin; sugar absent. There is an occasional red blood-cell in the microscopic field, but casts are found. There are a moderate number of leukocytes in each field.

Stomach Test.—The motor meal showed a normal emptying time. The Ewald meal showed free acid of 20, total acidity 35, occult blood absent. Pepsin and rennin present in normal amount.

Renal Functional Test.—Shows 75 c.c. the first two hours, with a total elimination of 45 per cent.

Stool shows some mucus and moderate amount of undigested food; no animal parasites were found. There is no occult blood.

Blood Wassermann test was negative.

x-Ray examination made by Dr. Jenkinson showed normal findings of both lungs except for a haziness of the base on both sides. The right heart measured 4 cm., the left heart 8.2 cm. The aortic arch measured 5.7 cm. Examination of both kidneys and ureters and urinary bladder did not show any evidence of stone. The esophagus showed a delay of the bismuth at the cardia, with a dilatation of the esophagus near the cardia. The sacculation had the appearance of a diverticulum above

the diaphragm. The diaphragm was in normal position on both sides. The excursion of the diaphragm was normal. Hernia of the stomach into the diaphragm could not be definitely excluded. The stomach was normal in size and shape. The pylorus was spastic, but on re-examination no evidence of spasm was found. The duodenal bulb was of normal size,
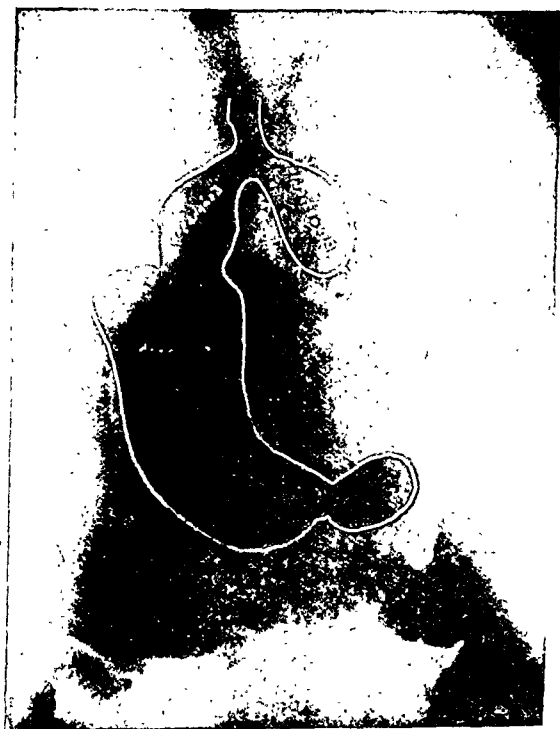
Fig. 19.

shape, and position. The stomach was empty at the end of four hours. The cecum was fixed. The colon filled rapidly and did not show any defects or obstruction. There was marked spasm throughout the colon. Dr. Jenkinson concluded that the patient had a diverticulum of the esophagus at its lowest position.

While the patient was in the hospital she suffered from one of her typical attacks. This was relieved by passing the stomach-tube. Four ounces of material were obtained, which was entirely undigested and did not show any free acids. This material was removed when the tube had gone down only part way, and evidently at once relieved the pressure upon her heart and cut short the attack.

The patient which you have just observed has evidence of a mass behind the heart, which by the opaque meal shows a definite connection with the esophagus and stomach. Although it is not definite that the mass seen in Fig. 19 is clearly a diverticulum of the lower end of the esophagus, yet the fluoroscopic evidence and likewise the laboratory evidence rather point to this view. It is possible that she has a hernia of the stomach which gives the impression of a diverticulum of the lower esophagus. I am inclined to the view, however, that she has a diverticulum of the lower esophagus and that it is at times overdistended, causing pressure upon the heart, and this, along with the gastro-intestinal upset, induces the marked depression and collapse which have been described. When the patient vomits this pressure is relieved and slowly the patient regains her equilibrium. Diverticula in this portion of the esophagus are uncommon. The patient's history is not typical for an ordinary diverticulum, which usually lies at the midportion or at the upper portion of the esophagus. The usual history of dysphagia is absent and one would not suspicion the presence of the diverticulum which the x-ray so clearly shows. It is only at times that the sac fails to empty itself and causes pressure symptoms that she gets her attacks. The patient herself has noted that when she is very careful in the selection of her food, eating mostly liquids and semisolids, that she does not have trouble, and that it is only when she takes the coarser foods that trouble may come on. This may explain her obstructive symptoms at intervals.

In esophageal diverticula the symptoms rarely develop before forty-five. These diverticula may attain considerable size. They must be differentiated from dilatations due to

spasms, strictures, and carcinoma. The type and shape of the dilatation as seen by the x-ray rules out a dilatation due to spasm, and the fact that the patient has intervals when she is perfectly free from symptoms speaks against organic stricture of any cause, as well as that due to carcinoma. The entire clinical picture and the course of the case is against a diagnosis of carcinoma. The only relief that the patient can have would be by the aid of surgical treatment. She will be referred for such advice.

SYPHILIS OF THE SPINE

Case II.—The next patient, referred by Dr. Rutledge, Mrs. M., aged sixty-one, married, entered the hospital on April 15, 1921, complaining of pain in the lower lumbar region, loss of weight, loss of appetite, insomnia, and progressive weakness.

Onset and Course.—The pain in the lower lumbar region has been present for the last ten months, and became considerably worse after an automobile accident in which the patient was severely jolted. At that time she says her head struck the top of the car. The pain is felt in the lower lumbar and upper sacral region. It was evident shortly after the accident. The pain was diffuse in the region described. The pain was not relieved by various measures which the patient tried, and in January, 1921 it became distinctly worse and more continuous. The only relief that the patient gets is by morphin. Movements of the spine aggravate the pain. The pain does not seem to have any relation to her food, nor is it relieved by bowel movements. Since January. 1921 the patient has lost about 40 pounds. She also complains of progressive weakness and loss of appetite. She tires very easily and has become very nervous and irritable. At times she has a marked generalized pruritus.

Past History.—She had the usual diseases of childhood and typhoid fever at the age of twelve.

Family History.—Her father died at the age of fifty-one of smallpox; mother died at eighty-nine of old age; one sister living and well, with no history of tuberculosis or carcinoma in the family.

Marital History.—Her husband is living and well, but has a positive luetic history. She had one miscarriage thirty-six years ago, but says this was an interrupted pregnancy. There were no other pregnancies.

Menstrual History.—She began to menstruate at fifteen, every twenty-eight days, lasting for three days. Her menopause began at the age of forty-five.

Physical examination reveals a rather poorly nourished white female, about sixty years of age, with blotchy pink skin, especially over the back and lower dorsal region, and over the inner aspects of the thighs. There was no tenderness over the mastoid, maxilla, or frontal regions. The nose is negative. The teeth are absent and the tonsils are atrophic. The eyes are normal in their reactions. There is a slight generalized superficial adenopathy. The thyroid gland is not enlarged. The chest is symmetric and expansion is good. Both lungs are hyper-resonant and no râles are to be heard. The heart is not en-larged. The tones are normal. The blood-pressure measured 120 systolic and 75 diastolic.

Abdomen.—The muscle tone is lost and there is no rigidity. There is generalized tenderness. The liver and spleen are normal.

Extremities.—Reflexes are normal throughout. There is loss of muscle tone.

Examination of the back was made by Dr. John L. Porter, who found that the entire lower dorsal and lumbar spine was stiff. No distinct exudate could be felt over the spine. Deep jarring caused slight pain, especially in the lower lumbar region. The patient gets out of bed with assistance, and walks more easily and with less pain than would seem possible from the history and x-ray findings. From the physical findings and the x-ray findings Dr. Porter's opinion was that both carcinoma and tuberculosis could be definitely excluded. Her complaint of pain and her moaning seemed to be partly due to the mental condition of the patient. He stated that there was a syphilitic involvement of the second and third lumbar vertebræ, as shown in Fig. 20.

Laboratory Findings.—The blood showed red blood-cells, 4,140,000; white blood cells, 7900; hemoglobin, 82 per cent. Differential white count was: small lymphocytes, 22; large lymphocytes, 5; polymorphonuclear eosinophils, 3; polymorpho-nuclear neutrophils, 69; basophils, 1.

Stool showed negative findings.

Urine showed a specific gravity of 1013, trace of albumin,

sugar absent. A few hyaline and granular casts were seen in each field, with a moderate number of leukocytes.

The blood Wassermann was negative.

The spinal fluid showed a cell count of 7. The colloidal gold reactions were not marked, but a reaction was seen as in syphilis, and the Wassermann test of the spinal fluid was a mild positive.

Fig. 20.

x-Ray examination was as follows: Left heart measured 7 cm.; right heart, 3.7 cm.; aorta, 5.9 cm. The posterior mediastinum is clear. The second and third lumbar vertebræ show a marked degree of osteosclerosis and the tissues are very dense and somewhat irregular. The articular surfaces are ragged.

The interpretation of the plates by Dr. Jenkinson was that the pathology was due to syphilis. x-Ray examination of the gastro-intestinal tract did not reveal any pathology.

Syphilis of the spine is a much more common condition than is ordinarily believed. In the literature, examinations of large groups of cases for syphilitic bone involvement showed that as much as 50 to 60 per cent. of the cases have syphilitic involvement of the spine. Often this is without the knowledge of the patient; further, this may occur without symptoms referable to the spine. Any of the joints or bones of the body may be involved and the pathologic changes are similar to those found in other parts of the body when the spine is involved. Commonly, involvement of the spine follows trauma, as was present in this case, but the spine may be involved without trauma. The spine is more or less rigid.

Whitney and Baldwin say that arthritic luetic lesions are of two types, either the syphilitic infection produces a lesion similar to that found in arthritis deformans, or the lesion may be of the toxic type. in which many joints are affected, and of these the spine is the most common. When the spine is affected it is more or less uniformly stiff and the x-ray shows definite evidence of trouble. The process attacks the synovial membranes of the various vertebræ, leading to lessened motion. At first the rigidity is due to spasm of the muscles of the back, but later, as the acuteness of the process subsides, adhesions form which limit motion and in many cases give complete fixation. These adhesions are not affected by antisyphilitic treatment. Usually three or four vertebræ are involved, but at times as many as six or eight may be affected. More rarely the entire dorsal region may be diseased and fixed. A large part of the backache of secondary syphilis is probably due to this cause. It is interesting to note that in many cases of spinal syphilis the blood Wassermann test is negative.

Tuberculosis gives the most trouble in the differential diagnosis, but the x-ray shows in syphilis an increase in tissue, whereas in tuberculosis it shows rarefied areas due to bone absorption.

MEDICAL MANAGEMENT OF DUODENAL ULCER

Case III.—Mr. E. D. C., aged thirty-one, entered the hospital on May 2d. He has the typical story of duodenal ulcer and has laboratory and definite x-ray evidence of a lesion in the duodenal bulb, which is undoubtedly a peptic ulcer. I am giving you merely a résumé of his history, for I wish to give you the details of the treatment which he will have during the entire course of the medical ulcer management.

There are a great many methods of medical ulcer treatment and each has its own advocates. Not until we shall know the exact cause of peptic ulcer will we have a specific remedy. Surgeons and internists are still each insisting that the best ulcer treatment belongs in their own spheres. I do not feel that a peptic ulcer should be treated surgically until definite surgical indications arise. If the ulcer fails to heal after a thorough and prolonged course of medical treatment, or if it shows any signs of perforation, then surgical aid should be sought. Likewise, if adhesions or strictures have formed which produce a high grade of motor insufficiency, or if repeated small, menacing hemorrhages occur, or if any signs of malignant change are present, I refer the cases to a surgeon. But unless these definite indications are present, I strongly urge the patient to take advantage of a prolonged course of hospital treatment, to be followed by a prolonged course of diet and medication for many months after leaving the hospital.

The ulcer treatment which I have followed for the past ten years is one that contains all of the good points of the various methods of treatment which have been advocated in the past. I first insist that every possible focal infection be carefully sought for and removed. This is especially true of diseased teeth and tonsils. Then the patient is put to bed and starts out with a modification of the Leube treatment. For a week or ten days nothing at all is given by mouth. The necessary fluids are supplied by retention enemata of glucose solution.

A quart is given every eight hours, and once during the day in the interval between these a thorough cleansing enema is given. Priessnitz compresses are kept on the abdomen constantly during the entire time. Then comes a period of one or two weeks of duodenal feeding of peptonized milk and peptonized eggs. Through the same tube orange juice and laxatives may be given. The feedings are given at two-hour intervals from 8 A. M. to 8 P. M. The amount of food is increased each day. When the tube is removed the modified Lenhartz treatment is started. Equal parts of milk and cream frozen as an ice-cream are given by mouth at two-hour intervals, the first day $\frac{1}{2}$ ounce, the next day 2 ounces, and the third day 4 ounces. These are followed in twenty minutes by an alkalinizing powder consisting of calcined magnesia and calcium carbonate. The dose is arranged according to the stomach analyses and symptoms of the patient. This powder is given the first few days in a small amount of ice-water, and the amount of water increased each day. On the fourth day the patient takes the milk and cream ice cold in larger amounts until 8 ounces are given every two hours. The amount of water with the powder is also increased. After a few days in place of the milk feeding a cream soup is given, and this is made without meat or meat stock and without any seasoning. Orange juice, cereals, and puréed vegetables are gradually added. Later, stewed fruits of all kinds, except berries, eggs, and toast, using unsalted butter freely, are also given. Frequent gastric analyses and examination of the feces are made to decide the progress of the patient.

During all of this time the patient remains quietly in bed, using the warm compresses constantly. This treatment continues for six weeks. At the end of that time, if no complications have come up, the patient is allowed to be out of bed and gradually takes up his ordinary duties. A program of three meals a day consisting of cereals, puréed vegetables, stewed or baked fruits, except berries, cream soups, toast with unsalted butter, orange juice, milk and cream, and eggs, is allowed, and between meals and at bedtime a glass of half-milk and half-cream is ordered. One-half hour after each feeding a

powder containing suitable doses of calcined magnesia and calcium carbonate are given, and one-half hour before the three meals a powder, consisting of neutralon and strontium bromid, to which frequently the extract of belladonna is added, is given. The dose of the various drugs is arranged according to the needs of the patient. This combination given before meals diminishes the amount of acid secreted by the stomach when the meal is taken, and I consider this of the greatest value in the control of the acid formed in the stomach. This, after all, is the greatest factor in preventing a relapse of the trouble.

The patient remains on this plan of treatment for six months or more, and then meat is gradually added to the diet. Stomach tests and stool examinations are carried out at stated intervals during this period, and later on an x-ray examination is made to decide more accurately just what progress has been made.

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CLINIC OF DR. JULIUS H. HESS

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL
FOR CHILDREN)

SPASMOPHILIA

DURING the past six weeks we have been seeing a group of
cases in our wards in numbers which would lead one to believe
that we are dealing with a condition appearing. in epidemic
form.   I refer to young infants ranging in age from four months
to two years, most of whom have entered the wards with a
diagnosis of acute infections with superimposed signs of active
spasmophilia.   The most striking feature lies in the fact that
during the early months of winter, notwithstanding the fact
that we had many infections of the upper respiratory tract and
the lungs, only at infrequent intervals preceding March 1st
have we seen these clinical manifestations on the part of the
nervous system.

CASE I

The first case which we will present is D. V., an Italian boy,
admitted March 15, 1921, at the age of six months.   His weight
is 13 pounds, 12 ounces.   He entered with a history of con-
vulsions, a marked rhinitis, and bloody stools, although more
recently constipation was present.   He had also shown a marked
muscular weakness and loss of weight covering a period of six
weeks.

The Onset and Course.—On February 1st he had severe
convulsions lasting fifteen minutes; three days later he had
nineteen convulsions during the day; following this he was
free from convulsions until March 14th, when he had three
more, and two more on the 15th.   In a description of the child's

general condition the mother gives a history·of typical carpopedal spasms and laryngeal stridor. The birth history is that of a full-term spontaneous delivery without postnatal complications. His early feeding history consisted of two months of breast feeding, at which time he was put upon condensed milk for about three months, and then upon whole cow's milk. Previous to the starting of the whole milk he had not been seriously ill, although he showed a general muscular weakness, made no attempts to sit up, and had a marked pallor.

Physical Examination.—Shows a fairly well-nourished infant, somewhat less than average weight for his age, not seemingly acutely ill, although he had had two convulsions on the day of entrance. His temperature was 100° F. per rectum, pulse 120, and respiration 30. His head is rather large, with prominent bosses, the anterior fontanel measures 1½ cm. in diameter, the posterior fontanel practically closed, and there is a moderate degree of craniotabes. There is a marked nasal discharge, with some excoriation; the throat is negative and he has no teeth. He has a cervical adenitis involving both superficial and deep chains and the glands are only moderately enlarged. The chest is funnel shaped with a moderate rosary and some flaring at the costochondral junction. The lungs and heart show no pathologic changes. The abdomen is greatly distended and tympanitic, but not tender. There is a large umbilical hernia and the stomach and intestines can be outlined through the abdominal wall. The liver is enlarged and palpable three fingerbreadths below the costal arch; the spleen cannot be felt. The upper extremities show marked deformities in that the hands are in a state of clonic carpal spasm, and about 1 inch above the wrist-joint there is a marked bilateral angular deformity, which to all appearances is either due to a marked bending or fracture of the bones of the forearms. The epiphyseal area of the wrists are markedly enlarged. The legs are in a state of clonic pedal spasm, with enlargements at all of the epiphyseal regions. The spine shows considerable scoliosis. His reflexes are all exaggerated and there is a bilateral Chvostek sign. He has a C. O. C. of 2 ma.

The laboratory findings were as follows:

Hb. 60.   R. B. C., 3,000,000.

W. B. C., 13,000.

Differential S. M.   L. N.   P.   T.   E.

35   28   35   1   1

Von Pirquet, 0.

Urine negative except for a few pus-cells.

The roentgenographic examination showed typical rachitic changes in both the upper and lower extremities and bilateral

Fig. 21.—Case I. D. V. Spasmophilia as a complication in a case of florid rickets. Bilateral pathologic fracture of both bones of the forearm.

pathologic fractures of both bones of the forearm (Fig. 21). Bismuth meal shows a markedly distended and dilated stomach (Fig. 22), with prolonged retention, some of the meal being present in the stomach after seven hours. The lower ileum is

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the cecum. The gastro-intestinal tract was practically empty after twenty-four hours (Fig. 23).

The dietetic treatment consisted of equal parts of milk and barley-water with 3 per cent. of sugar, six feedings of 6 ounces each. Ten grains of calcium lactate was given five

Fig. 22.—Case I. D. V. *Showing distended and dilated stomach. Prolonged retention, some of the meal being present in the stomach after seven hours.*

times daily, and phosphorized cod-liver oil, one teaspoonful twice daily, in orange juice. On the second day in the hospital repeated convulsions necessitated the use of chloral and bromids per rectum, following which he was put upon cereal gruels, vegetable broth, and human milk. On the twelfth day he was

again put upon a weak cow's milk formula, and his general condition has since improved. The carpopedal spasm disappeared on the sixth day in the hospital and his nasal infection gradually subsided.

Fig. 23.—Case 1. D. V. Showing marked dilatation of the lower ileum and, more particularly, the region of the cecum. Some retention in stomach after seven hours.

With the exception of bronchitis, which developed about two weeks after admission and which was associated with a high temperature and increased electric reaction, he has shown general improvement.

In reviewing this history several features are of interest. He developed his rickets while on condensed milk and his con-

vulsions while being fed on whole cow's milk shortly after the development of an upper respiratory tract infection. At the same time he also had a dyspepsia. Associated with his rickets was a history of chronic constipation, marked anemia, a dilated stomach and ileum. Following the development of his prolonged carpopedal spasm he suffered from a bilateral fracture of both bones in the forearm.

CASE II

The second case, B. R., age four months, that of an infant prematurely born at the eighth month, which was artificially fed almost from birth. He entered the hospital with a history of repeated convulsions. This infant also had an upper respiratory tract infection and a right-sided otitis. He is markedly rachitic, with a large open fontanel and extreme degree of craniotabes. The veins of the forehead and scalp are especially prominent. The mouth and throat are negative, there is a moderate degree of cervical adenopathy, the chest is pigeon breasted, shows an extreme degree of rosary and Harrison's groove. The lungs and heart are negative. The abdomen is distended and tympanitic and he has a large umbilical hernia. The liver is enlarged and displaced downward. The spleen is not palpable. The reflexes are all exaggerated and Chvostek's sign is present. He has a C. O. C. of 1 plus and the A. O. C. is 2 ma., while the A. C. C. is 3. Roentgenographic examination shows a moderate enlargement at the epiphyseal line, with a slight cupping and no visible epiphyseal nuclei (Fig. 24). The latter roentgenographic findings are typical for early rickets in premature infants, in contradistinction to the more marked findings of rachitic lesions in older full-term infants, in whom the cupping, flaring, and saw-toothed appearance represent the typical findings of the second stage of rickets. On breast milk and calcium lactate therapy the convulsions have ceased, the Chvostek has disappeared, and the electric reactions have approached the normal in five days.

Summarizing this case, we have the very early development of marked rickets in an artificially fed, prematurely born

infant, with an active spasmophilia at the fourth month of life, which is a period much earlier than similar conditions are seen in the full-term infant. The attack was probably precipitated by the intercurrent infection. The symptoms have rapidly disappeared following the institution of breast-milk feeding and calcium medication.

The term *spasmophilic diathesis*, as applied in the clinical sense, refers to a constitutional anomaly characterized by a general hyperexcitability and irritability of the nervous system. The most frequent active manifestations are general convulsions, laryngospasms, spasmodic apnea, and carpopedal spasm. Among the latent manifestations are Chvostek's facial phenomenon and Trousseau's sign. Among the most constant findings is Erb's sign, the presence of hyperexcitability of the peripheral nerves evidenced by reaction to the galvanic current. We will have occasion to refer to these clinical manifestations again.

Fig. 24.—Case II. B. R. Premature infant, age four months, with development of rickets and spasmophilia, showing moderate enlargement at the epiphyseal line, slight cupping, and no visible epiphyseal nuclei.

Several *etiologic* factors are of importance. The exact rôle of *heredity* and *familial predisposition* is open to question; however, it is our belief that not an inconsiderable number of infants developing active clinical manifestations are predisposed from birth. To this class of cases belong the restless, nervous infants with a tendency to pylorospasms and repeated vomiting. The colicky infant, which is often overfed to pacify it, thereby resulting in the development of nutritional disturb-

ances, belongs to this group. Added to the difficulties of these infants are the presence of neurotic parents who often show little inclination to train their infant. We must, however, not make the error of believing that all infants developing spasmophilia during their infancy have this hereditary tendency. The active manifestations are usually evidenced between the sixth month and the second year in the full-term infant, while in the premature they may be seen as early as the fourth month.

The *feeding* history, with its associated nutritional disturbances, is of great importance in the interpretation of the clinical manifestation. It is but rarely seen in the breast fed, and most frequently seen in the infants fed upon proprietary foods, more particularly those who have had repeated changes in their diet at the suggestion of kind friends. Overfeeding with cow's milk frequently aggravates the condition and may precipitate the active manifestation. We will again have occasion to refer to the rôle of diet when discussing the pathogenesis. One of the most striking phenomenon in the treatment of these cases is the rapid disappearance of all manifest signs when the infants are placed upon breast milk, with the reappearance upon the addition of relative excesses of whey or large quantities of cow's milk. It therefore becomes evident that the nutritional disturbances following quantitative and qualitative errors in diet are important factors in its development. One of the most frequent results of improperly balanced diet is the development of rickets, and in a very large majority of these infants active rickets can be demonstrated clinically and by roentgenographic studies. Corroborative evidence of the importance of the rôle played by rickets is the good effect to be noted from the addition of cod-liver oil, with or without phosphorus, to the diet. Premature infants show a marked tendency to the early development of florid rickets as well as complicating spasmophilia. It is especially true of this class of infants who have been artificially fed from early infancy. By far the greatest number of cases are seen during the winter and spring months, and are associated with some form of infection, more commonly

of the upper respiratory tract. So far as is known the vitamins play no specific part in its development.

PATHOGENESIS

Several theories have been advanced. All of these have as a basis some fundamental disturbance in metabolism. The most generally accepted has been based on experimental and clinical evidence of decreased calcium retention. More recently considerable data has been produced seemingly pointing to increased retention of the alkali phosphates (K and Na) with a secondary diminution of Ca salts as important factors. Perversions in parathyroid function because of the similarity of the tetany produced by pathologic changes and extirpation of these glands and the clinical manifestations of spasmophilia must be considered as a possible factor in its pathogenesis.

Calcium Metabolism.—A calcium deficiency in the tissues has been demonstrated by numerous investigators, more especially in the brain and blood. The earlier investigations on the blood have more recently been confirmed by Howland and Marriott,[1] who found the calcium of the blood-serum to be low in this condition, averaging 5.6 mg. per 100 c.c. of serum in a group of 18 cases, the lowest being 3.5 mg. per 100 c.c. of serum, the average normal amounts being 10 to 11 mg. per 100 c.c. They found a normal calcium content in the serum in convulsive disorders due to other causes. These same authors found the magnesium content of the serum to be within normal limits even in the presence of active spasmophilia. The relation of calcium to the symptoms of spasmophilia has been studied extensively, especially its influence on the electric excitability. Physiologists have shown that certain mineral ions exert a specific effect on muscle-nerve irritability. Rosenstern[2] and Sedgwick[3] reduced the electric irritability in spasmophilic infants by administering large doses of calcium by mouth. Loeb's[4] findings indicate that Na and K increases the threshold

[1] Howland and Marriott, Quarterly Jour. Med., Vol. XI, 1917–18, 289.
[2] Rosenstern, Jahrb. f. Kinderh., LXXII, 1910, 154.
[3] Sedgwick, J. P., St. Paul Med. Jour., 1912, Vol. XIV, 497–519.
[4] Loeb, J., Oppenheimer's Handbuch der Biochemie.

for excitation, while Ca and Mg tend to decrease this. This muscle nerve irritability is the function of the quotient $\dfrac{Ca+Mg}{Na+K}$, as designated by Reiss.[1] During a diarrhea Holt[2] has demonstrated there is a much greater loss of Na and K than Ca and Mg in the stools. Diuresis and catharsis often cause an improvement in the spasmophilic symptoms. Consequently, there is much clinical and experimental evidence that spasmophilia is much influenced by the relationship between the Ca-Mg. and Na-K group of ions.

Alkali Phosphate Metabolism.—More recently considerable work has been published on the results of observations on the influence of the alkali phosphates, more especially potassium and sodium diorthic phosphates, in normal infants and cases of latent and active spasmophilia. Howland and Marriott, finding the calcium of the serum greatly diminished when the inorganic phosphorus of the serum was high in severe nephritis with acidosis, studied the blood findings in spasmophilia to ascertain whether there was an accumulation of inorganic phosphates in this condition which might account for the reduction of the calcium. They answer the question in the negative after having determined the inorganic phosphorus in the serum in active tetany. They found the calcium low, but no significant increase in the phosphorus content. They consider 1 to 3.5 mg. per 100 c.c. of serum as the normal limits. In a group of cases they found the serum to contain 1.0, 3.0, 4.0, and 2.7 mg. in the presence of active manifestations.

Binger[3] was able to produce tetany by the intravenous injection of orthophosphates. He found not infrequently in conditions with a calcium deficiency alone that tetany was absent. These authors state that the reduction of calcium alone is not sufficient to bring about the symptoms of tetany unless certain other conditions are satisfied. The question

[1] Reiss, Ztschr. f. Kinderh., 1911, III, 1.
[2] Holt, Courtney, and Fales, Amer. Jour. Dis. Child., 1915, IX, 213.
[3] Binger, Jour. Pharmacol. and Exper. Therap., 10, 1917, 105.

as to the nature of such associated findings must be answered by further study.

Jeppsson and Klercker[1] in a series of experiments found that by the feeding of .20 gm. of P_2O_5 per kgm. body weight to normal infants and .10 to spasmophilic infants, they were able to produce symptoms similar to those seen in active spasmophilia, or to activate the manifestations in latent cases (.20 gr. of P_2O_5 represents in alkali diorthophosphates .27 gr. of K_2O and .18 gr. of NA_2O, or .49 gr. of K_2HPO_4 and .39 gr. of NA_2HPO_4). When using the potassium salts at times these results were manifested in a few hours. With the sodium salts larger quantities were required, and often the symptoms were not evident for two or three days. In rickets and spasmophilia in the brain, muscles, and bones it has frequently been noted that there is an increased storage of phosphorus, and at the same time the phosphorus excretion may be increased; therefore, even in the presence of an increased excretion, the tissues are excessively rich in phosphorus.

They believe that most of the spasmophilic children had received an excess of alkali phosphates, more especially during the first and second year, with the exception of the first few months of life, that is, during the period of predilection. Given an eight-month infant weighing 8 kgm., receiving 1 liter of cow's milk a day, it will therefore be fed .25 to 3 gr. P_2O_5 per kgm. This will not be received altogether as alkali phosphate, but a large proportion of it will be. By experimenting with children with latent spasmophilia they demonstrated that the whey caused less irritation when a considerable part of the phosphates was removed by precipitation.

These latter authors state that the importance of the PO_4H-ion cannot be laid to the lessening of the calcium content of the organism alone, although this may be an important action. The *parathyroid theory* is to a large extent based upon the fact that the physiologic and chemical findings in spasmophilia in infants and parathyroid tetany in animals are nearly identical. The histologic evidence is conflicting and for the most part.

[1] Jeppsson and Klercker, Zeitschr. f. Kinderh., 1921, Vol. 28, 71.

negative. Accidental removal of the parathyroid gland in humans and experimental excision of these glands in animals have both resulted in .a tetany that resembles in its clinical manifestations the spasmophilia of infants. Following the animal experiments Howland and Marriott[1] have demonstrated a diminution in the calcium content of the blood. These findings have been verified by MacCallum and his co-workers,[2] who also found a decreased calcium content in the brain and an increased excretion. They believe that the parathyroid gland regulates calcium metabolism and that failure in its secretion results in a lessened retention. Greenwald[3] in his experimental studies found that the phosphorus excretion in the urine of his animals was greatly decreased (to as low as 8 per cent. of the normal) shortly after operation, but after development of tetany it increased rapidly, occasionally to an amount in excess of the preoperative content. He also found an increase of the phosphorus content of the blood before the appearance of tetany. There was also a sodium and potassium retention. He believes that following the extirpation of the parathyroid there is a decreased excretion through the kidneys and an abnormal retention in the tissues of the alkali phosphates, which is followed by a decreased retention and an increased excretion through the kidneys as soon as the spasms develop. The great question which still remains unanswered is whether the clinical manifestations are due to the calcium decrease or to a positive poison with some other substance.

There is, however, great question as to the relationship of parathyroid dysfunction and tetany in the infant. Pathologic studies lead us to believe that parathyroid lesions in infantile tetany are the great exception. Parathyroid lesions have been described in patients who have shown no evidence during life of the pathognomonic findings of tetany.

In summarizing the pathogenesis we may state that a diminution of the calcium salts in all probability is the most important

[1] Howland and Marriott, Transactions Amer. Ped. Soc., 28, 1916, 200.
[2] MacCallum and Voegtlein, Jour. Exp. Med., 1909, XI, 118.
[3] Greenwald, J., Jour. Biol. Chem., Vol. 14, 1913, 370.

factor in the development of this condition. However, the possibility of an absolute or relative excess of the sodium and potassium salts, especially the phosphates, playing an important rôle cannot be overlooked. The relationship of disturbance in parathyroid functions to the diminution of calcium tissue content must be made the subject of further study before its importance can be fixed.

SYMPTOMS

You will recall that in defining the term "spasmophilic diathesis." as used in a clinical sense, a number of symptoms were mentioned which are considered pathognomonic of this condition. It would, however, be a great error, were you led to believe that the average case as seen in our wards presents all of either the manifest or, again, the latent signs of this symptom-complex. More commonly our diagnosis must be based on one or two of the characteristic signs. and therefore it would be well to review briefly the signs in their most frequent sequence. Erb's sign. better designated as increased electric hyperexcitability. Chvostek's facial phenomenon, and Trousseau's sign are thus enumerated in the order of their frequency. Of the manifest signs, generalized convulsions are the most frequently seen, while laryngospasm, spastic apnea, and carpopedal spasms are of less frequent occurrence. It therefore becomes obvious that many cases of spasmophilia would be overlooked were it not for the uncovering of the latent manifestations during the course of a routine physical examination. On the other hand. the interpretation of convulsions must be dependent to a large extent upon a careful study of the clinical history. Otherwise cases of spasmophilia will be overlooked, or the diagnosis of this condition made in cases due to other causative factors.

Erb's Sign (Increased Electric Irritability).—The test is made with a galvanic current. and for this purpose a small battery made up of dry cells answers the purpose best for the finer tests. However, there are batteries with transformer attachments which can be used with ordinary lighting current. A large flat electrode (2 inches in diameter) is placed on the

chest or upper abdomen of the infant and a small Stintzing electrode 2 or 3 cm. in diameter is placed over the median nerve just below the elbow or over the peroneal nerve below the popliteal space. For the purpose of making the test the opening contractions are used. The C. O. C. is usually first used because of its being the easier of the two opening contractions to demonstrate, due to the fact that in normal infants under two years of age approximately 9 ma. are required to obtain a reaction. After completing this test the pole should be reversed and the A. O. C. tested. In making these tests the C. C. C. is of little value and the A. C. C. is only used for comparison with the A. O. C. The test, whether studying the C. O. C. or A. O. C., should always be begun with a current of sufficient strength to produce a contraction of the muscles of the fingers and the thenar and hypothenar groups of muscles, when the median nerve is used, and the muscles of the toes and plantar surface of the foot when the popliteal nerve is used.

The average normal reaction under two years of age will approximate the following:

C. C. C.	A. C. C.	A. O. C.	C. O. C.
2	3	5	9

In children under five years of age the following electric reactions are diagnostic of spasmophilia:

C. O. C. under 5 ma.

A. O. C. with less current than that causing an A. C. C. and under 5 ma. up to the end of the second year. After this age there is a normal tendency for the A. O. C. to appear with less than 5 ma. A C. O. C. with less than 2 ma. is indicative of the appearance of early convulsions, and should always be considered an indication for active treatment.

Chvostek's facial sign depends upon the hyperexcitability of the facial nerve. Tapping lightly with the finger along the course of the nerve midway between the zygoma and the angle of the mouth results in a contraction of the ali of the nostril, angle of the mouth, and in most cases the inner canthus of the eye and the eyebrows. The appearance of a Chvostek phenome-

non under two years in the absence of birth trauma indicates tetany. After three years the Chvostek phenomenon is not infrequently found in milder grades in apparently normal children.

Trousseau's sign is elicited by moderately compressing the nerves and vessels of the arm by the hand, or an elastic constriction is evidenced when present by the development of a carpal spasm.

Tetany is characterized by arthrogryposis or carpopedal spasms, which are seen clinically as tonic spasms of the hands and feet. The fingers are usually flexed at the metacarpophalangeal joints and the phalanges extended; the thumbs are adducted almost to the little finger; the wrist is flexed at an acute angle, and the whole hand drawn somewhat to the ulnar side. If the spasm is very marked, no motion is allowed at the wrist, but movements at the elbow and shoulder are usually normal. The feet are strongly extended, sometimes in the position of typical equinovarus, and the first phalanges of the toes are flexed. If these conditions persist for a long time, edema of the dorsal surfaces of both hands and feet will develop. The clonic contractions remain sometimes for hours, and even days, and are evidently quite painful.

Convulsions (Eclampsia).—Evidences by loss of consciousness, spasms of the face and extremities, at first tonic, later clonic. They usually last only a few minutes—may or may not recur. In exceptional cases a status eclampticus develops. More commonly, however, in the milder types the infant will recover from the individual attack quickly and without seeming after-effects, thereby resembling the petit mal attacks of epilepsy. Only in the severest forms do they resemble the grand mal attacks, and only rarely is coma seen following the seizure. Due to the fact that spasmophilia is most frequently seen during the active period of early dentition and that many of the cases show delayed dentition the laity are inclined to ascribe teething erroneously as a cause.

Laryngismus Stridulus (Inspiratory Laryngospasm).—This may occur without provocation or following crying or fright.

There is an inspiratory crow due to spasmodic closure of the larynx, associated with cyanosis, and it may be followed by convulsions. The condition may persist over a period of several weeks or until proper treatment is instituted.

Spastic Apnea or Expiratory Apnea.—This may result in cardiac death.

COURSE

The acute symptoms may last from a few days to several weeks. It varies in most cases directly with the treatment. Occasionally a persistent type is met with. Rickets, nutritional disturbances, and infections must be overcome.

PROGNOSIS

Acute Attack.—It should always be guarded, as it depends upon the condition which accounts for the symptoms. The convulsions add to the gravity of the condition, as also does the presence of thymus enlargement.

Pertussis often is of grave import as a complication. In the majority of cases the prognosis is, on the whole, good.

After-life.—While many of these children show no after-effects, a considerable number suffer from nervous manifestations in later life, as headaches, pavor nocturnus, enuresis, tic, stuttering, etc. Others show mental retardation.

TREATMENT

Dietetic.—*Latent spasmophilia* should be treated prophylactically to prevent the development of the manifest symptoms. As spasmophilia is relatively rare in *breast-fed* infants, the latent spasmophilic on artificial diet should be fed human milk if it can be obtained. When the condition develops in breast-fed babies, breast milk from another source may be tried if available; however, improvement will usually be slow until a mixed diet and proper therapeutic measures are instituted. If the latent phenomena recur when cow's milk is again fed, these infants should be fed human milk for a considerable time. When this is impossible, the cow's milk should be limited to a pint a day, or it may be replaced by albumen (Eiweiss) milk which

has a low whey content. A carbohydrate, cereal, and vegetable diet should be instituted whenever the infant's age permits. Fruit juices are also essential. It is often well to keep the diet as small in quantity as is compatible with progress in the child. Sodium and potassium salts should be avoided. A certain group of *artificially fed* infants will do better when cow's milk in all forms is excluded from the diet. In such cases a mixed carbohydrate diet, consisting of cereals and sugars, together with vegetable purées and fruit juices, should be instituted whenever possible.

They should not be kept for too long a time on a strict cereal diet due to the danger of development of marasmus (flour injury). It is often well to keep the diet as small in quantity as is compatible with the progress of the child. A temporary stationary weight should not be cause for concern.

Medicinal.—Cod-liver oil alone or combined with phosphorus offers the best form of medicinal treatment during the latent stage; however, in the presence of active manifestations they must be supplemented by other forms of treatment.

The calcium salts in the form of chlorid, lactate, or bromid may be used to advantage both during the active and latent stages. The calcium salts should always be prescribed in solution to prevent injury to the mucous membranes. Calcium chlorid or calcium lactate may be administered in 0.5 to 1 gm. doses three or four times daily, or calcium bromid in one-half these amounts.

Convulsions.—An initial does of castor oil or magma magnesia is a valuable adjunct to the further treatment in the absence of marked gastro-intestinal irritation. A short period of starvation diet, consisting of tea and saccharin, should be followed by human milk where possible. When it is necessary to feed artificially the infant should be kept for twenty-four hours following the short starvation period on cereal gruel plus sugar, this to be followed by low milk and carbohydrate or albumen milk. Following this the previously recommended dietetic treatment may be instituted. It may be necessary to control the active manifestations by bromids per mouth

and chloral hydrate per rectum, and when the temperature is high, saline enemata, packs, sponging, and baths are indicated. In the presence of repeated convulsions we have frequently seen excellent results following the administration of 10 c.c. of an 8 per cent. solution of magnesium sulphate hypodermically daily for one, two, or three days.

Further treatment should include the secondary anemia, and not infrequently a change of climate is of advantage so that an outdoor life can be lived. Under all circumstances a good hygienic and dietetic régime should be instituted.

Efforts should be directed toward the prevention of all respiratory infections, more especially during the winter and spring months.

Parathyroid feeding has shown no results.

CLINIC OF DR. PETER BASSOE

PRESBYTERIAN HOSPITAL

ENDOCRINE GROWTH DISTURBANCE—ACROMEGALY, GIGANTISM, DWARFISM

THIS morning I desire to call your attention to certain gross disturbances of growth and development dependent on endocrine disorders, and in which the principal and apparently primary disturbance is found in the hypophysis. However, it has been demonstrated that disturbances of many ductless glands, alone or in combination, may lead to disorders of growth, but the most striking ones are those dependent on hypophyseal disease. Normal ossification and proportionate growth above all depend upon normal development and balanced physiologic activity on the part of the hypophysis, thyroid, and gonads. It has been said that the momentum of growth present in the normal fetus at birth will cause it to reach a size normal at seven or eight years, and for further growth in length to occur the hormone of the hypophysis is of particular importance. If this is lacking, as in cases of teratoma or congenital cyst of the hypophysis, we get a case of so-called hypophyseal dwarfism in which there is also arrest of development of the gonads, so sexual maturity is not attained and ossification is delayed, the epiphyseal lines often remaining open far into adult life or even to old age. The condition of failure to attain puberty and normal epiphyseal closure, which is associated with the persistence of other characteristics of childhood, is known as *infantilism*. Stunting of growth is a common but not an essential feature of infantilism. In fact, I shall point out later that it may be present in cases of excessive growth—even in true gigantism. Not only the hypophysis, but the thyroid and the gonads are important factors in the production of infantilism.

To realize the importance of the thyroid in connection with growth it is only necessary to recall to you the stunted growth of the typical cretin and the startling growth stimulated in cretins by thyroid opotherapy. The influence of the gonads on growth and development is demonstrated by the profound changes brought about when these structures become functionally active, a stage which we designate puberty. For an understanding of our subject it is of the greatest importance to consider the modifications of growth which are the result of failure of genital development as seen in individuals castrated early in life (eunuchs). This condition has been studied particularly by Tandler and Grosz and others, in the "Skoptzi," members of a cult residing chiefly in Russia and Roumania. Like capons, their extremities are disproportionally long. The head is small, the sella turcica relatively large, which is indicative of compensatory enlargement of the hypophysis. The thyroid is small, while the thymus may persist beyond the normal period. The growth of hair on the head is thick, but beard and pubic hair are lacking. The larynx is small and the voice does not change at the usual age. In gonad deficiency due to congenital tumors of the testicle a hyperactivation of the hypophysis may occur, resulting in an extremely rapid growth, as in the famous case of Sacchi, of a boy who also was sexually mature at nine years. As *eunuchoidism*, first described by Griffith, we designate a developmental disorder from defect of the testicular interstitial glands, in which the characters described in eunuchs also are present. When there is an additional tendency to excessive deposits of fat we have reason to suspect hypophyseal disorder as well, while carious teeth and marked skin changes implicate the thyroid.

Case I.—This overgrown boy of fifteen years, already 6 feet, 2 inches tall, is brought to the clinic on account of his defective mental development and alarming growth. He has gained 5 inches in height during the past year. His mother states that he was born several weeks before term, but nevertheless he weighed 9 pounds at birth. His birth was normal and early development normal. He walked at one year. He

has done very poorly in school, and in spite of regular attendance, he has only reached the fourth grade. You will note that his head is small, its circumference being 53 cm., that the extremities are long in proportion to the trunk, that his pelvis is very broad and heavily cushioned with fat so as to give it a feminine appearance. Pubic hair is present, but with the sharp
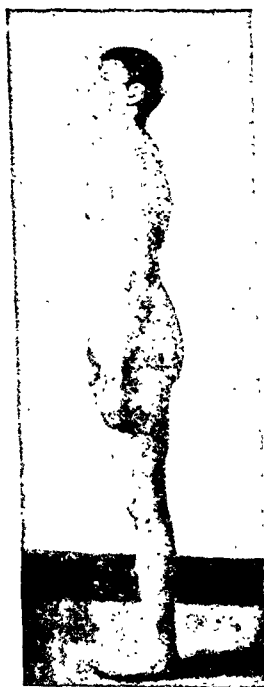
Fig. 25.—Case 1. Infantile gigantism in boy of fifteen years. Note small head and long limbs.
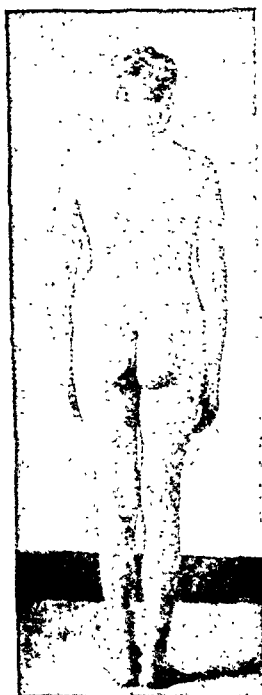
Fig. 26.—Case I. Posterior view of patient shown in Fig. 25.

horizontal demarcation above characteristic of the female. The testicles are very small; the thyroid also is small. Muscular development and strength are good. Roentgenogram of the head reveals open sutures. The greatest anteroposterior diameter of the sella turcica is 1 cm.; its depth, 7 mm.

We look upon this case as essentially one of eunuchoidism

with associated polyglandular disturbances principally affecting
the hypophysis.

Case II.—This boy of seventeen years is 6 feet, 3½ inches
tall. His father was a giant who measured 7 feet, 8¾ inches,
and 3 of his brothers are taller than himself. His extremities
are also disproportionately long, and the height of his iliac
crests is 126 cm. or 60.75 per cent. of his total height. He, too,
has a rather small head, very small genitals, marked feminine

Fig. 27.—Case II.   Infantile male giant, seventeen years old.

type of pelvis, and large pendulous breasts. It is interesting
that every month for two or three days he has pain in the flanks
and hips and flushing of the face, but no blood appears any-
where. There has been no sign of development of sexual func-
tion. We have not had an opportunity to obtain a roentgeno-
gram of the head.

Having now fixed in our minds the chief features of the
excessive body growth mainly dependent on eunuchoidism, we

are prepared to appreciate the essentially different picture presented in cases of frank hypophyseal hyperfunction.

Case III.—This large man of forty-five years presents a striking contrast to the young men just shown. He is no taller and not of symmetric build, but the distribution of the excessive growth in him is a different one. The length of the trunk and extremities is proportionate. His head is large, its circumference 61.5 cm. Most striking is the prominence and massiveness of the lower jaw, the prominence of the nose and superciliary ridges, and the thickness and roughness of the skin. Next to the prominence of the facial part of the skull our attention is directed to the disproportionate size of the hands and feet. The former measure 21.5 cm. from the wrist to the tip of the middle finger, and the latter are 27.6 cm. long. The circumference of the hands, 25 cm. of the right and 23.5 cm. of the left, is also excessive. Roentgenologic examination shows that the broadness of the hands and feet is largely due to thickening of the subcutaneous tissues. A roentgenogram of the head brings out excessive width and depth (each 2 cm.) of the sella turcica, large frontal sinuses, and the elongation and projection of the lower jaw.

The appearance of this man is absolutely pathognomonic. Whatever else there may be the matter with him, he certainly must be a case of acromegaly. Let us now go a little further into his history and complaints. He came to me a couple of years ago, sent by a physician who had given him antisyphilitic treatment for several months. He gave a history of having had a chancre at the age of twenty years and of having had three negative and two positive Wassermann tests. He was aware of his growth disturbance. He had to wear a larger hat and to increase the size of his shoes from 11 to 12½, and of his gloves from 9¾ to 10½. He was not aware of any impairment of vision, but with correction the best vision obtainable is 20/50 in the right eye and 20/30 in the left. No definite narrowing of the visual fields or change in the disk are noted. The pupillary and other reflexes have been normal. The Wassermann test with the blood when I first saw him was

positive, with the spinal fluid negative. The latter gave a cell count of 18 and a negative globulin test. I have given him considerable specific treatment, and the Wassermann test with both blood and spinal fluid has lately been negative. His urine at first was normal, but of late sugar frequently has been found, and occasionally a trace of acetone. What is the possible connection between his acromegaly, syphilis, and glycosuria? The syphilis we consider merely incidental, as experience has shown that whether acromegalic patients have had syphilis or not, postmortem examination invariably reveals adenoma of the anterior lobe of the hypophysis, a tumor which is of the same character whether syphilis happens to have been present or not. The glycosuria in this case undoubtedly is related to the acromegaly and not to the syphilis. In 176 cases of acromegaly Borchardt found 63 complicated with diabetes and 8 with alimentary glycosuria. Labbé has pointed out that when acromegaly and diabetes coexist the former appears first, and that polyuria is likely to persist after the sugar has disappeared. He is probably right in his assumption that the diabetes of acromegalics is not due to associated disease of the pancreas.[1]

Case IV.—This robust Greek gentleman of forty-one years came to us a few days ago not because of feeling ill, but to find out whether it is safe for him to marry. He had a chancre and secondaries fifteen years ago, received treatment promptly by his physician, Dr. Papageorge, and no further symptoms of syphilis have been noted. Enlargement of the head, especially the lower jaw, and of the hands and feet appeared at about the time he was treated for syphilis, and Dr. Papageorge thinks there has been little change since that time. He is very strong, feels well, and has no disturbance of vision. The Wassermann test is now negative with both blood and spinal fluid. You

[1] This patient later died of lobar pneumonia. A necropsy was held, and the most interesting points in the anatomic diagnosis were the following: hyperplasia of the hypophysis and the sella turcica, enlargement of liver and spleen, hyperemia of the medullary portions of the adrenals, lobar pneumonia of right lower lobe, and right fibrinous pleuritis. Histologic examination showed the usual type of hypophyseal adenoma; no gross or microscopic signs of syphilis.

will note his typical acromegalic facies, with prominent superciliary ridges, broadened nose, and prominent lower jaw. The tongue is very broad. Roentgenogram of the head shows no sellar enlargement, but the frontal sinuses are enlarged.

The absence of sellar enlargement does not exclude acromegaly, as adenoma of the anterior lobe of the hypophysis with hyperplasia of the chromophil cells may exist without enlargement of the gland, as has been demonstrated by Dean Lewis.

This is an instance of arrested disease. It is not uncommon, as has been shown in a large series of cases by Schlesinger, to have a brief period of hyperphyseal overactivity in youth and to have the morphologic changes remain as permanent results of this brief disturbance.

Case V.—A married Jewish woman forty years old, the mother of 2 children and previously well, has been complaining of quite severe intermittent occipital pain for four months. She has menstruated only once in the last two years, and then very slightly. A year and a half ago a physician had observed her for a time and found her blood-pressure to range between 180 and 195. Since then she has been treated for pyorrhea. Her blood-pressure now is only 135. She tells us that her ophthalmologist found her eyes normal two months ago.

Looking at her, you will note that although she is heavily built and stout—she weighs 170 pounds—her nose is extremely broad and the nostrils wide and her hands and feet are broad. On the other hand, the lower jaw and superciliary ridges are not prominent. On questioning her we learn that while she wore No. 6 shoes three years ago, she now wears No. 8, and she has to wear No. 8 gloves instead of 6¾, as formerly. The pupils are normal. As you see me test her visual fields roughly with my fingers you will note that the temporal fields seem slightly narrowed. On ophthalmoscopic examination the disks appear a little pale on the temporal side, which suggests the possibility of a beginning atrophy, which has not yet caused any demonstrable impairment of her central vision. The roentgenogram of the head shows an enlarged, oval sella turcica

measuring 15 mm. in width and 12 mm. in depth. Another feature which may be significant is that her skin has become rather dark, especially on the arms and trunk.

This case of undoubted early acromegaly in a woman emphasizes two important points about this disease: first, that amenorrhea is an early and frequent feature. The uterus, ovaries, and breasts undergo early involution, while the clitoris and to some extent the vagina tend to enlarge. Second, in acromegaly there is an early tendency to exaggeration of the male secondary sexual characters, and for those of the female to assume a male type. The female patient is prone to develop an increased and diffuse growth of hair, even the whole chest and breasts may be covered with hair, and the pubic hair assumes masculine outline. The pelvis gradually acquires the male shape, and with the increasing roughness of the skin, general coarseness of the features, and the hairy growth, the whole appearance becomes strikingly masculine.

Case VI.—The appearance of the next patient, a farmer twenty-eight years old, is so striking that you will have made your diagnosis at a glance. First of all, his size brands him as a giant. He is 6 feet, 8¾ inches tall and weighs 241 pounds. His features and the huge size of his hands and feet brand him as also acromegalic. In other words, this is a case of acromegalic gigantism. Let us now consider his case in detail. He tells us that he began to grow rapidly at the age of fourteen years and measured 6 feet, 5 inches at nineteen years. Since the age of twenty he has grown relatively slowly in height, but has broadened out and gained 50 pounds in weight. He has always been strong and served seven months in the army during the recent war. It is rather amusing that neither draft boards nor the camp examiners realized that his growth was pathologic, as he would have been rejected in that case. He is not aware of any impairment of vision, but he has been subject to frontal headache for the past five years. He tells us that his sexual desire and power were normal until five years ago, and since then have gradually diminished. You will note that his superciliary ridges and lower jaw are very prominent,

that his ears are large, that the tongue has large papillæ, and is large, measuring 6.5 cm. in width, that he has an abundant growth of hair, and that the genitalia are well developed. Let me give you a few measurements. As stated, his total height is 6 feet, $8\frac{3}{4}$ inches, or 205.5 cm.

His height at the iliac crests is 126.5 cm. The circumference of the head is 62 cm., the pupillary distance 7.5 cm., the width of the nose 4.5 cm. The circumference of the hands is 25.5 cm. Both middle fingers are 12.8 cm. long, 3.4 cm. wide, and the nails are 2 cm. in width. The feet are 32 cm. long. As you compare his hands and feet with their roentgenograms you realize that the great increase in width is due to enlargement of the soft parts. The larynx is prominent and the thyroid is slightly enlarged. Although he does not complain of his eyes, perimetric examination shows an appreciable narrowing of the fields in all directions both for form and colors. The urine obtained when he entered the hospital a few days ago was free from sugar, but ingestion of 50 gm. of glucose caused sugar to be present in six specimens voided during the following twelve hours. Roentgenogram of the head shows decided enlargement of the sella turcica and of all accessory sinuses.
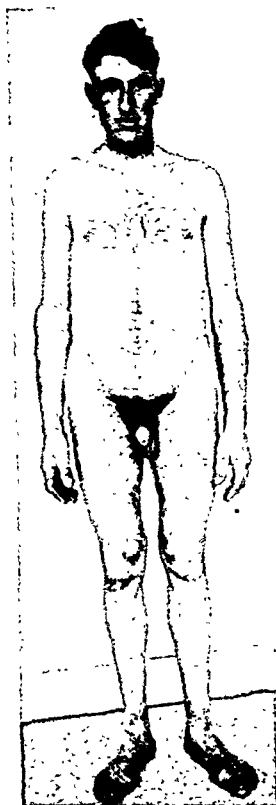
Fig. 28.—Case VI. Acromegalic gigantism.

The Relation Between Acromegaly and Gigantism.—Comparing this man with the two young boys first shown, who

are giants still in the making, let me lay down some rules which are rather dogmatic and schematic, but in a general way true:

1. There are no "normal" giants.

2. All cases of gigantism belong in one of the following three groups: (a) gigantism with infantilism, (b) gigantism with acromegaly, (c) gigantism with infantilism and acromegaly.

3. When gigantism and acromegaly are combined the former condition appears first.

Fig. 29.—Case VI. Feet of acromegalic giant.

4. About half of all reported giants have had acromegaly.

5. Both gigantism and acromegaly are due to hyperfunction of the hypophysis. In a general way it may be said that when the gland disorder begins in childhood or early youth a giant is produced, while if the disease commences after the epiphyseal lines have closed growth can only take place at the tips of the segments ("acrons"), and acromegaly results.

Further Remarks on Acromegaly.—In presenting the patients I took particular pains to point out the most essential symptoms

in connection with the abnormal stimulation of growth in certain directions. These are the primary glandular symptoms. We have also subordinate glandular symptoms due to secondary or associated disorders of the other endocrine glands, such as amenorrhea and other genital symptoms, and abnormal pigmentation, growth of hair, and other skin changes attributable to the adrenals and thyroid. Then the enlargement of the hypophysis *per se* gives rise to two sets of symptoms: (1) local

Fig. 30.—Advanced acromegaly. (Case of Dr. Dean Lewis.)

pressure symptoms chiefly affecting the optic chiasm and nerves, causing changes in the visual fields, and frequently optic atrophy and blindness; (2) in later stages, if the hypophyseal tumor grows to a large size, the usual general symptoms of brain tumors.

The Skeletal Changes.—I am, fortunately, able to show you the entire skeleton of a male acromegalic who was a patient in the service of Dr. Dean Lewis in the hospital for a long time. As you will see from his photograph (Fig. 30) the facial changes,

Fig. 31.—Skeleton of acromegalic patient shown in Fig. 30. (Kindness of Dr. Dean Lewis.)

roughening of the skin, and broadening of the hands were extreme. His sella was enormous and he had been blind for many years

before death. The necropsy revealed an unusually large hypophyseal adenoma, extensive bone erosion, and almost complete destruction of the chiasma. An interesting and rather common feature was the general enlargement of the viscera (splanchnomegaly). You see that the skull as a whole is large and thick, · and that the thickening of the cranial bones is due to the diploë, while the tables are thin. The sella turcica measures 36 mm. anteroposteriorly. Its floor is entirely destroyed, so it is continuous with the sphenoid sinus. The anterior clinoid processes are lengthened and sharpened, while the middle and posterior ones are destroyed. There are numerous wart-like excrescences of very spongy bone about the margins of the sella. The elongation of the lower jaw is very marked and its prominence is increased by the widening of the angle between the ramus and body. General features of the entire skeleton are: thickening and roughening of all bony points giving attachment to muscles or tendons; ossification of cartilages and ligaments, notably those of the spinal column, so as to cause fusion of vertebral bodies and processes in places; evidence of arthritis deformans in all joints of the extremities: erosion and destruction of articular cartilages, lipping of articular margins, and hyperostosis at the points of capsular and ligamentous insertion.

Prognosis and Treatment of Acromegaly.—Authors generally distinguish a rapid and severe form fatal in a few years, the ordinary chronic form fatal in eight to thirty years, and the benign form which does not shorten life. As previously stated, the process may be active for a brief period only and the condition remain stationary during the rest of the patient's life. In such cases the age of one hundred years is known to have been reached. There is little good to say about the curative effect of either glandular extracts or other drugs. We believe we are dealing with hyperfunction of the hypophysis and, consequently, can expect only harm from administration of this gland during the active stage. In late stages, however, as has been pointed out by Cushing, secondary hypopituitarism may ensue, and then hypophysis substance would be indicated.

Roentgenologic treatment appears to have been useful in

many cases. It has been particularly developed by Béclère, of Paris, who devised a method of passing the rays through the frontal and temporal bones so as to focus on the sella turcica. He and others report cases with not only subjective improvement, but actual widening of the fields of vision.

The surgical treatment has now attained a place which demands its consideration in all but the arrested cases. Two modes of approach to the hypophysis have been perfected—one through the nasal fossæ and sphenoid sinus, developed especially by Schloffer, Kanavel, and Cushing; the other, through the anterior fossa of the skull by means of a large flap. Krause, Frazier, and Adson have developed and described this method which has the great advantage of a wider exposure and a free view of the field.

Dwarfism.—A consideration of dwarfism helps to fix in our minds the principles so far laid down, and tends to some extent to confirm the views expressed. We may classify dwarfs as follows:

I. Proportionate Dwarfs:
    A. Primordial dwarfs (essential microsomia).
    B. Hypophyseal dwarfs.
II. Disproportionate Dwarfs:
    A. Achondroplasia (chondrodystrophia fetalis).
    B. Stunting of growth from rickets or Pott's disease.
    C. Cretinism.
    D. Congenital syphilis.

So far as the primordial dwarfs are concerned, we are at a loss for an explanation. This condition is usually hereditary and always present at birth. No endocrine or other abnormality has so far been demonstrated. These individuals merely appear like normal people seen through the large end of a telescope. A while ago I denied the existence of "normal" giants, but so far must admit that "normal" dwarfs apparently exist. The hypophyseal dwarfs are most interesting to us, as in all essentials they are the antithesis of acromegalic giants. They are small, but proportionate, have too little hypophysis tissue, do not acquire normal sexual or osseous development.

Of the "disproportionate" dwarfs I only wish to say a little about the victims of achondroplasia, or the "short-limbed dwarfs." The condition of relative shortness of the extremities is established early in fetal life and maintains about the same proportion throughout life. There are abnormal conditions at the epiphyseal lines, and many, but not oversuccessful, attempts have been made to explain the entire state by tightness of the amnion and other purely mechanical factors. There
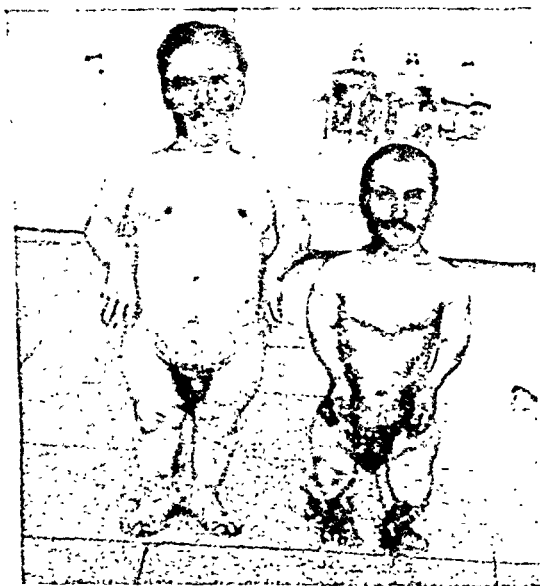
Fig. 32.—Achondroplastic dwarfs. (After Parhon and Shunda, Nouv. Iconogr. de la Salpêtrière, May–June, 1913.)

must be a common cause, as all of these achondroplastic dwarfs are remarkably alike. They have relatively large and broad heads and are remarkably strong and agile. Most of the dwarf acrobats in circuses belong to this class. Their prowess was known to the ancients, as a still existing statue of a dwarf gladiator from the time of Emperor Domitian shows him to be of this class. A feature of this statue is the penis, which actually reaches to the ankle. This suggests both a fact and a theory.

namely, the fact that in nearly all descriptions of these dwarfs the large size and excellent functional conditions of the genitalia are dwelt upon, and logically the theory that perhaps the dwarfing of these individuals is due to premature inhibition of cartilaginous growth by excessive gonad hormones. This genital theory actually has been advocated, most recently by a German writer, G. A. Wagner. He points out that achondroplastic dwarfs in many respects are the opposite of castrated or eunuchoid individuals by virtue of their short limbs, large heads, excessive muscular and genital development, strongly marked secondary sexual characteristics, and early ossification. Be this as it may, the mere theory helps in impressing on you the rôle of the endocrines in growth disturbances which I have tried to bring home to you.

It may seem to you a waste of time to devote much attention to these rare conditions which may impress you principally as mere curiosities without practical importance. Let me only remind you that it largely was scientific interest in the apparently equally unpromising and unimportant cretins which led to the epochal discovery of the importance of thyroid function, which discovery has opened an entirely new field in medicine of almost limitless possibilities, both in pathology and therapy.

CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

THE ADMINISTRATION OF DIGITALIS IN THE PRESENCE OF CERTAIN ACUTE INFECTIONS

GENTLEMEN: I should like to discuss with you this morning certain features of the administration of digitalis with which I have been particularly impressed during the past year. These impressions relate to the use of digitalis in certain of the so-called acute respiratory or influenzal infections. Several of these have been of the streptococcus variety. Out of a rather large number of such acute infections I wish to present to you 3 cases, as they summarize rather well most of the experiences of the others. Then we shall try, perhaps, to draw certain general conclusions applicable to this type of disease.

The use of digitalis in acute respiratory infections is, of course, not new, the most notable and wide-spread example being its use in the treatment of acute lobar pneumonia. Within recent years the use of digitalis in this disease has become practically a routine with the entire profession, and while I do not desire in any sense to convey the impression that such use is not warranted. I feel that there may be certain dangers and disadvantages in this routine procedure which, perhaps, are not thoroughly appreciated. I think there is no question that in the past the use of digitalis in severe cases of lobar pneumonia, particularly in the aged, in the presence of auricular fibrillation, or with definite evidence of heart failure, has been a distinct contribution to our management of this dread disease. During recent years, however, I have gradually come to question somewhat the routine use of digitalis in unselected cases of pneumonia in younger individuals, or in individuals without

signs of heart failure. Although during the war, in both the first and second streptococcus pneumonia epidemics and later in the influenza epidemic, I, in company with many others, ordered digitalis practically as a routine, and in large quantities in the treatment of these acutely ill soldiers, I must confess that in retrospect, I remember little or no evidence to show that it was of actual value. In fact, I am wondering just a bit whether there might not have been some harmful effects from its administration.

The pharmacology of digitalis has in recent years been rather completely and exactly worked out, both in experimental animals and in man, thanks to the work of Cushny, Hatcher and Eggleston, Cohn, Robinson, Pardee, and others. As a result of their work the exact mode of pharmacologic action has been made clear, namely, the effects of digitalis on the medulla, on the vagus, on the sino-auricular and auriculoventricular nodes, on the bundle of His and its branches, on the heart muscle directly, much of which evidence may be demonstrated by the electrocardiograph. Clinically likewise, the physiologic and toxic effects of the drug have been studied, the effects on hearts beating regularly, on hearts beating irregularly, in auricular flutter, and especially in auricular fibrillation.

One of the most frequent results of complete physiologic or mild toxic amounts of digitalis, so-called complete digitalization, is the production of partial or complete auriculoventricular heart-block. Associated with such heart-block one usually finds other evidence of digitalis-poisoning, namely, nausea, vomiting, headache, ringing in the ears, etc., which symptoms, together with the bradycardia ·(heart-block), promptly disappear upon the discontinuance of the drug.

While digitalis rather regularly in full physiologic dosage will produce heart-block, there are many other agents and conditions which either experimentally or clinically will produce a similar and practically indistinguishable effect; Lewis has listed these as follows: asphyxia, adrenalin, strophanthin, aconitin, muscarin, physostigmin, nicotin, glyoxylic acid, morphin, potassium salts, diphtheria toxin.

Parallel with the above list, which under certain conditions will produce heart-block, certain acute infections have been found which may result in the same fashion. A year ago in the American Journal of Medical Sciences I[1] published a series of 6 cases of postinfluenzal myocardial conditions in which either the auricle or conduction pathways of the heart were involved. In this article I referred to some work of Cockayne[2] on heart-block following influenza, wherein he reported 15 cases of partial block occurring in young men suffering from mild influenzal pneumonia. No mention was made of digitalis. Recently McCulloch,[3] in studying the effects of diphtheria on the heart, found 19 cases of a group of 80, which showed evidence of cardiac disturbance, a number of which showed complete or partial auriculoventricular dissociation as well as involvement of branches of the His bundle. In a more recent communication concerning the administration of digitalis to children with diphtheria McCulloch[4] pointed out that in his belief digitalis was definitely contraindicated, inasmuch as, because of the striking similarity of the effects of digitalis and diphtheria toxin upon heart structure, there would result a summation of the effects in the same heart. A rather similar effect I believe I have found in influenza and other allied respiratory (streptococcus) infections.

Without further discussion I should like to present to you my first case. This case concerns a young schoolboy, Joe M., aged thirteen, whom I saw in consultation with Dr. Jampolis in October last year. The boy's younger brother, a month before I saw him, had a mild case of scarlet fever, from which he made an uneventful recovery. About three weeks later Joe developed a sore throat and was seen by Dr. Jampolis' assistant, who diagnosed an acute follicular tonsillitis, and believed he could rule out scarlet fever. A throat smear showed a practically pure culture of a hemolytic streptococcus. About the sixth or seventh night of this illness, shortly after midnight, Joe was suddenly awakened from his sleep by a feeling of extreme illness; he felt weak and nauseated, and was barely able to reach the bathroom, where he vomited several times and then fainted.

His parents found him on the bathroom floor, felt that he was extremely ill, largely because of his pallor and weakness, and hurriedly summoned their physician. On his arrival Dr. Jampolis found the boy propped up in bed, breathing with considerable difficulty, ashy pale except for a moderate cyanosis of lips and finger-nails. His pulse was extremely slow, regular, and forceful, beating at the rate of 28 beats per minute. I saw him about eight hours later, during which time his condition remained practically unchanged. His pulse then was still 28, the intensity of cyanosis varied with his respiration, the venous pulse above the clavicle on the right side appeared to be about 120 to the minute. The heart tones were slow, regular, forceful, especially the first tone—free from murmurs. Except for a feeling of extreme prostration and weakness and some precordial oppression the boy appeared comfortable. I diagnosed a three-to-one postinfectious (streptococcus) heart-block, and because of the bradycardia and chest oppression gave him $\frac{1}{120}$ grain of atropin sulphate hypodermically. This resulted in no change in the heart rate, but did give definite relief from the substernal pressure and a general feeling of improvement. His condition remaining unchanged, during the next hour it was thought wise to transfer him to the hospital for more careful study. He entered about four hours later, an electrocardiographic tracing being made at the laboratory on his way to his room.

Fig. 33 shows the three leads of this electrocardiograph, showing, as you see, complete auriculoventricular dissociation and right bundle branch block. The boy was put at complete rest in bed with an ice-bag to his precordium and was kept completely quiet and isolated. No food or drink was allowed for twenty-four hours, and his only medication consisted of $\frac{1}{120}$ grain of atropin hypodermically every eight hours. Forty-eight hours later the complete auriculoventricular block had changed to a two-to-one partial heart-block (Fig. 34), six days later to a four-to-three block, and on November 11th, two weeks after the onset of his acute illness, the curves showed practically normal cardia mechanism. A week later he left

the hospital essentially normal. To summarize, then, this case is another example of an acute postinfectious (streptococcus) myocarditis involving particularly the bundle of His and its branches, resulting in a transitory complete auriculoventricular dissociation and right bundle branch block.

The second case which I wish to discuss with you is in contrast to the foregoing insofar that although it has also to do with heart-block, it is one produced by digitalis and not by an acute infection. This patient, Fred F., aged fourteen, I first saw
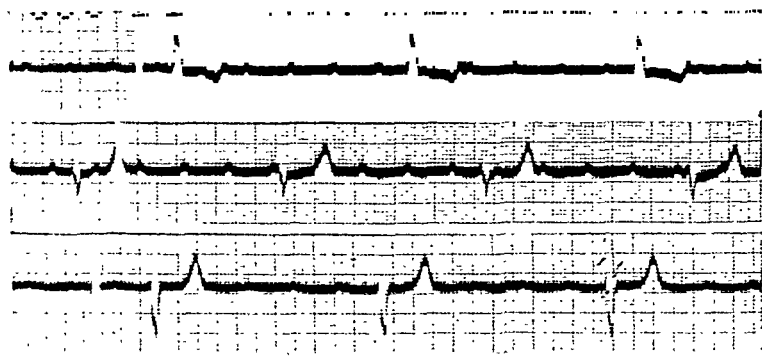
*J.M. C.I.II.III. oct. 27ᵗʰ 1920 · on entering Hospital
complete auriculo-ventricular block right bundle branch block*

Fig. 33.—Electrocardiograph (three leads—I, II, III) of young boy (J. M.), showing complete heart-block, result of an acute postinfectious myocarditis involving bundle of His.

in consultation with Dr. George Scupham, of Homewood, Illinois, in December, 1920. Fred had apparently been entirely well until eight months ago, at which time he developed an acute polyarticular rheumatism involving practically all the joints of his body. Although he was kept in bed at this time between two and a half and three months, he began to develop some dyspnea on exertion shortly after being allowed to get up. Three weeks before I saw him his ankles, legs, and abdomen began to swell, his dyspnea grew worse, he had sleepless nights, and vomited after practically every meal. Examination showed

a pale, thin boy, emaciated except for the swollen legs and abdomen, with a rapid weak pulse, and with cardiac dulness
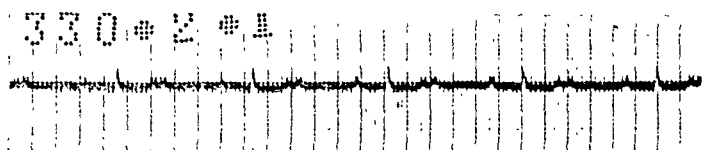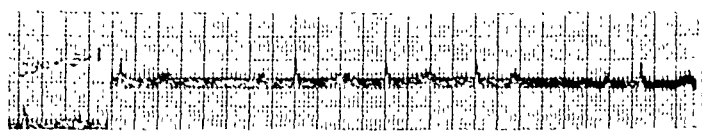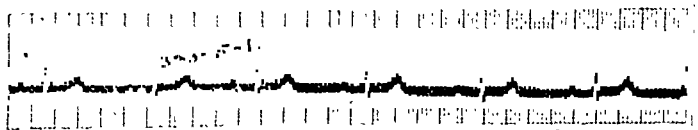
Fig. 34.—Electrocardiographs (single leads) taken on different days of same patient (J. M.), showing gradual recession of infectious heart-block, with, finally, restoration of normal sinus rhythm.

markedly increased in all directions, with a systolic and late diastolic murmur at base and apex, his condition becoming progressively worse under moderate amounts of digitalis. I

recommended more drastic measures for him at his home, but within a few days, owing to the difficulties of carrying out this
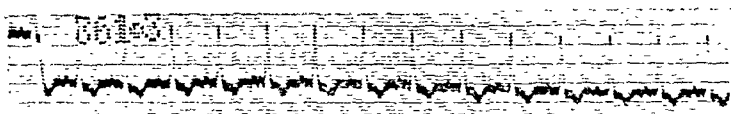
F.F. Dec.13" L.3. on entering hospital. advanced heart failure. no digitalis right preponderance. inverted T. 3
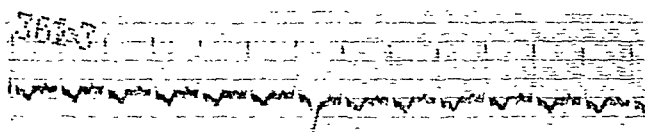
F.F. Dec.21" L.3 After 24 C.C. Tinct. digitalis. heart compensated .
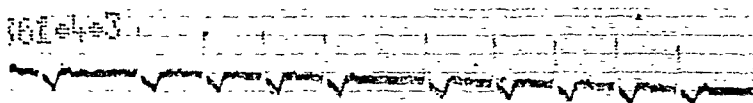
F.F. Dec 23 9.30 L.3. After 28 c.c. Tinct. dig. mild digitalis poisoning. Partial heart block. T wave negative deep and peaked
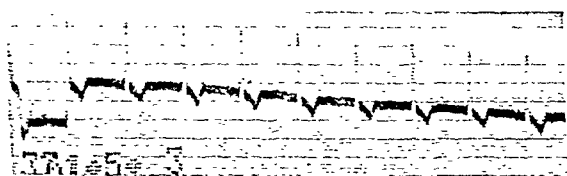
F.F. Jan.4" 921 L.3. digitalis discontinued normal cardiac mechanism

Fig. 35.—Electrocardiographs (single leads) taken on different days of young boy (Fred. F.) with advanced heart failure, showing production of partial heart-block the result of full digitalis dosage; with restoration of normal cardiac mechanism upon discontinuing drug.

régime at home, as well as the continued downward course of this disease, he was transferred to the hospital.

The first tracing of Fig. 35 shows Lead 3 of his electrocardiogram upon entrance to the hospital. During the suc-

ceeding ten days under complete isolation and bed rest, thora-
centesis abdominis, with the removal of 3 liters of ascitic fluid,
a Karell diet, limitation of fluids, and digitalis in full doses,
this boy's compensation rather quickly returned, with the
production on December 23d of a partial auriculoventricular
heart-block (third tracing, Fig. 35). Ten days later, with a
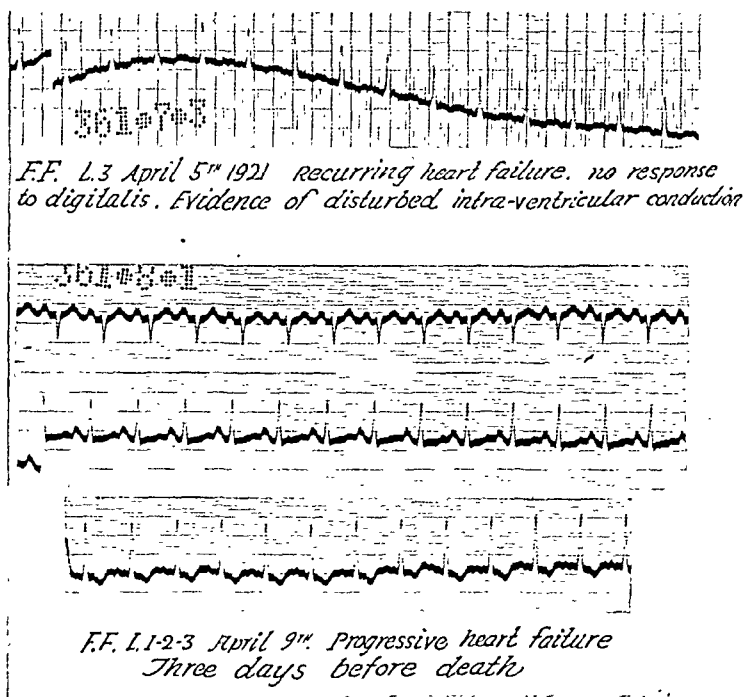
Fig. 36.—Fred F. (same patient as Fig. 35) three months later, with re-
current progressive heart failure, not responsive to digitalis, without heart-
block, with death from ventricular fibrillation.

discontinuance of the digitalis, the partial block had entirely
disappeared (fourth tracing, Fig. 35).

Fred returned home about four weeks thereafter with his
heart entirely compensated, not taking digitalis, with marked
improvement in his general health, and with instructions to
have his tonsils, which were evidently diseased and apparently

the portal of entry of his rheumatic infection, removed as soon as his condition permitted. However, after a three of four weeks' period at home, he again developed fever, sore throat and joint pains, and shortly thereafter evidence of a recurrence of his heart failure. He entered the hospital for the second time about the first of April, but in spite of practically identical management failed to improve. Tracing 1, Fig. 36 shows Lead 3 of an electrocardiogram taken April 5th, showing a failure of response to digitalis, and with some evidence of disturbed intraventricular conduction as shown by the slight notching of the apex of the R. wave and increased width of its bases. In spite of various other measures, namely, caffein, theobromin, various diet modifications, etc., his condition grew progressively worse, with death occurring from ventricular fibrillation, April 12, 1921.

This case, then, can be summarized as a postrheumatic pancarditis resulting in advanced heart failure, responding temporarily to digitalis, with the production of digitalis block, with subsequent recurrence of heart failure, refractory to digitalis, ending in death. In comparison and contrast to the first case it concerned itself likewise with a postinfectious heart condition, rheumatic, not streptococcal, far more severe and wide-spread, with heart-block a result of digitalis, similar to the infectious block, resulting in death.

Case III.—The third case that I wish to report to you briefly is perhaps the most interesting of all. It concerns itself with a woman, Leah L., aged forty-six, a housekeeper, for the courtesy of whose report I am indebted to Dr. M. L. Goodkind, on whose hospital service it occurred. Because of the difficulties with which this patient speaks and understands English and because of the fact that she was treated for several days in the Dispensary before entering the hospital, the exact details of the beginning of her illness are not clear. As far as I can make out she became ill about February 1st with an acute fever, right-sided chest pain, dizziness, weakness, sore mouth and throat, which conditions were variously diagnosed pneumonia, acute respiratory infection, mouth and throat infection, etc.

She was apparently given some tincture of digitalis, the exact amount I am unable to ascertain, but after four doses of these "black drops" she felt very much worse and thought she was going to "collapse." She entered the hospital February 4,
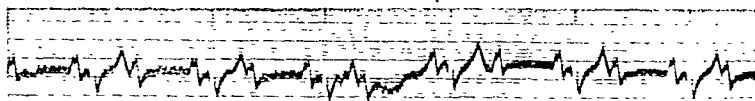
L.L. L.II Feb. 5 1921 on entering hospital 2-1 partial heart block P high - wide - bifurcate.

L.L. L.II Feb. 5-1921 after atropin
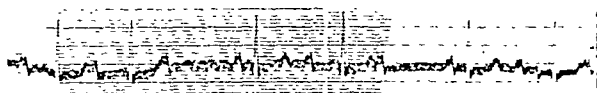
L.L. L.II Feb. 7ᵗʰ 1921 3-2 partial heart block bigeminal pulse
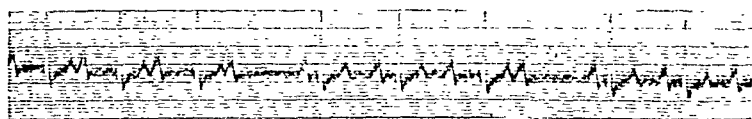
L.L. L.II. Feb. 8. 1921 4-3 partial heart block trigeminal pulse

Fig. 37.—Single leads from middle-aged woman (Leah L.) suffering from partial heart-block, the result of the administration of moderate amounts of tincture of digitalis in the presence of an acute respiratory infection; a summation, digitalis—infectious heart-block.

1921 in a semicomatose, at times actively delirious, condition, entirely disoriented, talking with great difficulty and irrationally, with a temperature of 100.4° F. and a slow, regular pulse of 40 beats per minute. Chest was negative. There was a systolic-

diastolic murmur at the apex, the urine contained much albumin and a few granular casts. An electrocardiogram made the following day, her pulse remaining slow, showed a two-to-one partial heart-block, the P wave being high, wide, and bifurcate (tracing 1, Fig. 37). Atropin was ineffectual. Two days later, with a general clinical improvement, she developed a typical bigeminal pulse, the result of a three-to-two partial
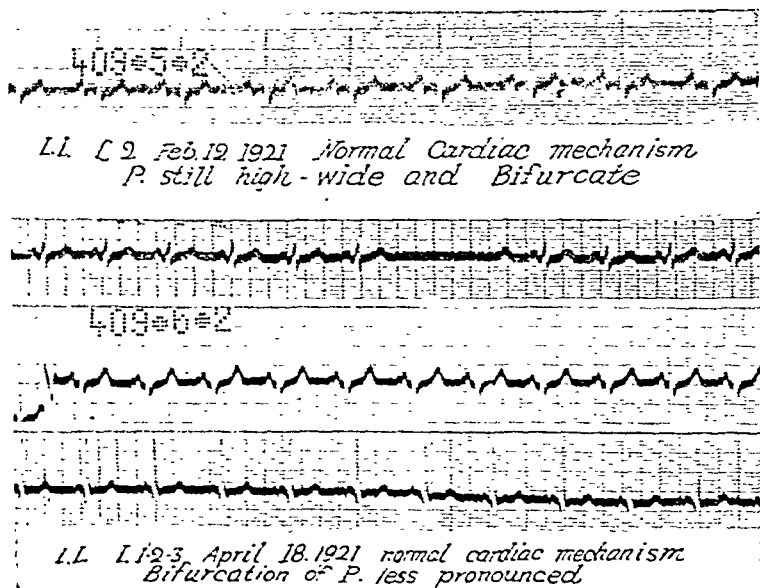
Fig. 38.—Leah L. (same patient as Fig. 37), showing prompt restoration of normal cardiac mechanism with discontinuance of digitalis and subsidence of acute infection.

block, the following day a trigeminal pulse from a four-to-three heart block, and four days later a normal cardiomechanism, with the exception of the anomalous P wave (tracing 1, Fig. 38). On leaving the hospital February 18th her heart was essentially normal.

It is difficult to conclude definitely about this patient, but I feel that in all likelihood it is a case showing a summation of the effects of digitalis and an acute infectious process, prob-

ably streptococcus. I judge this from the small amount of
digitalis she received, which, in the presence of an acute fever,
resulted in heart-block, and from which she was apparently
made worse, the curious appearance of the P wave, the bizarre
type of throat, and lung infection, etc. Of course, the crucial
test of this case would be the rate of the heart and the appear-
ance of the electrocardiogram before digitalis was administered,
but failing in this information, one is justified, I believe, in con-
cluding that this case should be put down as one showing a post-
infectious myocarditis involving particularly the auricles and
conduction pathways of the heart with a summation digitalis
effect resulting in partial heart-block.

In other words, although one can at this time have no way
of knowing the exact condition of the heart prior to the adminis-
tration of digitalis, it seems fair to conclude that this rather
unusual type of infection prepared or sensitized the heart in
some way so that small amounts of digitalis subsequently were
effective in producing this unusual picture of heart-block.

It may be profitable to speculate briefly regarding just which
factor in this type of infection is responsible for the production
of heart-block. In attempting to analyze this question it
would seem that either the fever *per se* or the toxic agent, or
both are responsible. Against the first possibility is the fact
that in the treatment of a large series of infections with high
fever of various kinds, with or without digitalis, heart-block is
a rather unusual incident. For example, Cohn,[5] in 49 cases
of lobar pneumonia *to which digitalis was given in physiologic
amounts*, found heart-block only 7 times, and much less fre-
quently, if at all, when digitalis was not administered. Further,
in many cases of prolonged fever, such as typhoid, malaria,
sepsis, etc., the occurrence of heart-block is quite exceptional.
Contrast with this the relative frequency of this interesting
cardiac condition in diphtheria, influenza, streptococcus infec-
tions, etc. One must conclude, therefore, that the involvement
of the conduction pathways of the heart is related to the specific
toxin or antigen of these three infections, rather than to the
general effects of fever.

In this connection, however, one may be permitted to call attention to some interesting work of Gunn[6] in 1914 on the isolated rabbit's heart, wherein he showed that strophanthus acts more quickly as the temperature of the animal is raised. Gunn believed that the quicker rate of flow through the coronary vessels at the higher temperatures was probably sufficient to account for this. In 1920 Hirschfelder, Bicek, Kircera and Henson,[7] experimenting on frogs and cats, showed that the lethal dose of digitalis in cats, whose temperature was raised to 43° C., is much smaller than the lethal dose at normal temperatures. They concluded that although the heart muscle was free from injury due to prolonged fever or toxemia, the fact of high temperature was alone sufficient to render it much more susceptible to the effects of digitalis than is the normal heart muscle.

CONCLUSIONS

What practical conclusion may one draw from this work? They may be stated briefly as follows: In the treatment of *atypical* broncho- and lobar pneumonia; influenza; streptococcus, and other mixed streptococcus infections; endemic, epidemic, or pandemic respiratory infections; digitalis and related digitalis bodies are probably contraindicated, or, if used at all, should be given in *small* doses and only in case of actually threatened or present heart failure, bearing in mind constantly during its administration the possibility of heart-block. If block occurs, atropin should be given at once in moderate amounts, repeated as necessary. If digitalis *must* be used, it probably never should be administered in the massive doses of Eggleston, as most certainly with such large amounts cardiac damage from digitalis summation can be predicted.

Just a final word regarding the wide-spread, routine use of digitalis in the presence of *frank* (pneumococcus) lobar pneumonia, typhoid, sepsis, and other types of prolonged high fever. Although the evidence in this group of cases is not so clear, I believe it is a fair statement that although it is possible that digitalis in such cases *may* protect the heart muscle and relieve it to some extent of an undesirable strain, one must not forget

that digitalis itself is essentially a toxic agent, and may be productive of more harm than we know. My feeling at the present time is that except in the aged, or in the presence of a rapid heart with auricular fibrillation, or in patients with distinct signs of heart failure, it is better practice to withhold the drug until its real need becomes apparent, at which time, by the use of large amounts if necessary, the heart can be completely digitalized in from twelve to thirty-six hours.

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CLINIC OF DR. JAMES G. CARR

Cook County Hospital

SCURVY

"Weariness, independent of exertion, heaviness of the body, dyspnea, especially upon moving about, putrefaction of the gums, foul breath, frequent loss of blood from the nostrils, difficulty in walking. The legs sometimes swell, sometimes waste, with livid, leaden-colored yellow or violet-colored spots. The face is generally of a sallow paleness." Such were the words in which Sydenham described scurvy. As illustrating this great clinician's power of observation and independence of spirit it may be added that Sydenham was one of the few eminent medical men of his time who insisted on the comparative rarity of scurvy. For at least two centuries this historic disease was looked upon as very common. The first accurate description was the work of the French historian Joinville, who told of the ravages of the disease in the sixth crusade which was led by Louis IX of France. From the time of the discovery of America, with the enthusiasm for exploration, and the long voyages attendant thereupon, scurvy became a well-known and much dreaded disease. Gradually the name was used to cover various diseases, until many physicians could see a scorbutic tendency in almost every disorder. In 1752 a monograph was published by Lind, which is regarded as the first classical and complete description of the disease. Nevertheless, scurvy was carelessly diagnosed for a long time thereafter; thus, Trosseau in describing "diphtheria of the mouth" (in which description he gives a very good picture of Vincent's angina) told of an outbreak in 1818 in a French Legion; the condition was diagnosed by the army surgeons as "land scurvy"; Trosseau accu-

rately points out that all the symptoms of scurvy, save the stomatitis, were lacking.

The presentation of a disease, known so long yet in these days regarded as so infrequent as to be virtually obsolete, may call for an explanation. Perhaps the most convincing reason for calling your attention to the disease is the very fact that it is not obsolete. The case before you is the fourth case of scurvy we have had in our own ward since last March. There were 18 cases of adult scurvy treated in this hospital last year. Perhaps we need to emphasize the importance of thinking about scurvy; a "forgotten" disease is likely to be overlooked. Comby, not many years since, made the statement that of 55 cases of infantile scurvy which he had encountered, 45 had been incorrectly diagnosed; the children had usually been treated for rheumatism, acute poliomyelitis, syphilis, or acute osteomyelitis. Yet a further reason may be offered for a discussion of scurvy; it is a type of the so-called "deficiency diseases," which have proved of so much importance during the past seven years, and which have still a great significance for us all.

The man whom we present today is thirty-four years of age; he was admitted to the hospital January 3, 1921; he was born in Poland, is married and lives at home, and has worked as a laborer. His mouth and gums became sore and tender about three months ago; his gums are better than they have been, though they are still very tender and bleed easily. He has been obliged for some time to eat only soup and soft foods. It appears that he has consulted a dentist, who, it would seem, had no thought of scurvy. He complains of a pain in the small of the back, which is worse when he first gets up. He has not been able to work for two months on account of pain and weakness in his legs; within the past week he has become unable to walk, says his legs are too painful and weak to support him. His feet are swollen. He does not complain of dizziness or of palpitation. Prior to one month ago he ate meat once or twice a week; he has had no meat during the past month. Aside from bananas, he rarely eats fruit. He eats potatoes once or twice a week, but the principal elements of his diet are soup,

bread, cheese, butter, and cake. Lately he has been constipated. He has noticed bloody urine at times. His personal and family histories are negative. He is very pale, anemic, and unable to sit up in bed.

Physical examination shows the presence of pyorrhea; the gums are spongy and bleed easily; they are ulcerated in places and very sensitive. The tongue is heavily coated; the breath is very foul. Chest and abdomen are negative. About the left elbow there is pain and tenderness, with limitation of motion in all directions. There are some discolorations over both thighs posteriorly. The calves of the legs are both swollen, firm, and very tender. The right knee cannot be straightened; it is swollen and tender, fluid is probably present. The ankles are swollen and tender, and areas of discoloration are present over both, over the dorsal surfaces of the feet, and are scattered over the legs. The feet are swollen, tender, and pit on pressure. The reflexes are generally exaggerated; no abnormal reflexes are present. Several specimens of urine were normal except for the presence of a trace of blood in the first one examined. The blood count showed hemoglobin, 34; erythrocytes, 3,000,000; leukocytes, 11,750; of the latter, 74 per cent. were polymorphonuclears. The Wassermann was negative. x-Ray examination of the elbow-joint showed no changes. His temperature has been up to 99.4° F. twice; otherwise it has been normal. His pulse is usually under 90, never over 100.

Before we undertake a discussion of the subject of scurvy, I want to go over with you the histories of 2 patients who were presented to the Senior Class last year.

M. M., age fifty-three, of Irish birth, a janitor by occupation, was admitted to the hospital April 20, 1920. He complained of pain in the right knee, of sore gums, foul breath, loose teeth, general weakness, loss of weight, and reddish spots about the knees and over his body. For some six months his diet had consisted mainly of coffee, bread, and cakes (bakery products of various kinds), with practically no fresh vegetables or meat. The pain in the knees began ten days prior to admission; six days later he noticed that they were swollen, and the reddish

spots appeared. The gums had been sore for ten days. His previous history was negative, but an item not to be overlooked was the fact that for some time he had lived alone and boarded himself.

On physical examination the teeth were found to be generally carious; many were loose; the gums were spongy, with granulating, fungous-like tissue. There was a small ulcer on the right edge of the tongue. The heart, lungs, and abdomen showed no abnormalities. Distinct generalized myoidema was noted. The right knee was swollen, but not tender. There were purpuric spots about both knees, both ankles, and along the calves of both legs. The reflexes generally were normal, though the patellar and Achilles' reflexes were noted as active. Examination of the blood showed hemoglobin, 50 per cent.; erythrocytes, 3,360,000. The urine was normal, the Wassermann reaction negative. There was no fever at any time. Three weeks after admission the patient was discharged in good condition.

L. M., fifty-one years of age, also a janitor by occupation, was admitted March 27, 1920. The symptoms on admission were pain, swelling and discoloration of the left thigh and leg; there was also discoloration of the right thigh. Further symptoms were general weakness, hemorrhage from the gums, and paralysis of the left side of the face. Prior to admission he was not well for seven weeks. The facial paralysis was the symptom of onset, followed the next day by pain and tenderness about the left knee; this involvement of the extremity grew worse, and was later associated with marked discoloration. There was a history of syphilis, dating back thirty years. After the onset of the symptoms mentioned he received four injections of neosalvarsan; these were given after the blood was examined, and a report made that the result was positive for syphilis. Under this treatment the patient was no better, rather he grew worse, and became greatly dissatisfied because of his own failure to interest his physician in regard to his sore gums; the patient's frequent insistence that something be done for his sore mouth was ignored. The patient was an unmarried

man, who boarded himself, eating almost no fruit or vegetables, but mainly a diet of meat, beans, and macaroni; much of his food would be kept for two or three days after cooking, and then eaten.

On physical examination the facial paralysis of the left side was obvious; all the branches of the nerve were involved. The teeth were carious; the gums were swollen and bled easily. The whole left lower extremity was greatly enlarged, pitting on pressure; posteriorly there was a purplish discoloration from the hip to the ankles; the tissues were hard and brawny. There were many purpuric areas on the extensor surfaces of both lower extremities. The reflexes were normal. The blood-pressure was 130/100. There was albumin in the urine, but no blood or casts. The Wassermann test gave a negative reaction. As mentioned, he had been treated with neosalvarsan. x-Ray examination showed no evidence of subperiosteal hemorrhages. At no time was there fever.

In this last case there were two features worthy of special mention: (1) the history of the treatment which he had received for syphilis; (2) the facial paralysis.

There is much to be said in regard to the interpretation of a positive Wassermann reaction. The mere presence of a positive reaction does not warrant us in attributing whatever symptoms may be present to the syphilitic infection. We have seen several typical cases of lobar pneumonia in which this reaction was positive, yet the course of the disease was not unusual, nor did it seem wise to attempt specific treatment during the course of the pneumonia. A patient with a positive "Wassermann," just as a patient with tuberculosis, or chronic arthritis or any disease of long duration, may develop another disease, essentially independent of the chronic condition; too much reliance on the laboratory test may easily lead to error. In the case under discussion the patient was given four injections of neosalvarsan because he had a positive Wassermann; there was a good bit to be said in favor of the opinion that an active syphilis was present; the history, the facial paralysis, and the positive reaction made a suggestive picture. But the Wasser-

mann did not account for the scorbutic symptoms, and an open
mind would have appreciated that aggravation of the symptoms
in the face of specific treatment needed an explanation. The
truth is, that too often we allow our minds to be warped by a
report from the laboratory; once the laboratory says positive
"Wassermann" or "Widal," we are too prone to excuse our-
selves from any further effort to explain all the findings which
a case may present. Neither a positive Wassermann, nor a
Widal, nor the presence of tubercle bacilli in the sputum make
it impossible for an individual to have, as the cause of manifest
symptoms, a stone in the kidney, or an epidemic meningitis,
or a scurvy. Too much reliance must not be placed on labora-
tory results, valuable and indispensable as these aids have be-
come. Clinical experience, skill, and judgment have not been
superseded; the diagnosis should account for the findings
present; it must not be determined on the basis of laboratory
work alone.
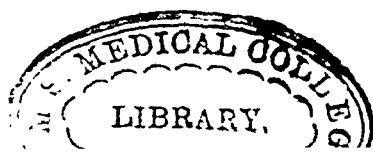
The facial paralysis was of unusual interest. Three possi-
bilities presented themselves as etiologic factors: (1) syphilis,
(2) infection, causing the condition known as Bell's palsy, (3)
hemorrhage into the nerve or about it. Bell's palsy cannot be
proved or disproved. It is not a plausible diagnosis in the
presence of definite etiologic factors; in this connection more
than that is hardly warranted. Syphilis is often associated
with facial paralysis; however, against the syphilitic nature of
this particular paralysis we had three considerations: (1) syph-
ilitic facial paralysis is a secondary rather than a tertiary mani-
festation; (2) after the patient's admission to the hospital the
Wassermann reaction was negative. This was not surprising
in view of the history given by the patient of specific treatment;
but if the facial paralysis was specific, then the Wassermann
reaction was "cleaned up" without any simultaneous improve-
ment of the paralysis; (3) the paralysis was the symptom of
onset, but was followed almost immediately by the joint symp-
toms of scurvy; you will recall that the patient stated that the
joint involvement followed the facial paralysis "the next day."

I have made no attempt to review the literature, but I

have consulted some standard text-books and systems, and I
have found no mention of facial paralysis as a complication of
scurvy. Bierich, whose report on 1343 cases we will refer to
later, does not mention facial paralysis. Nevertheless, in view
of the reasons cited above as evidence against the syphilitic
nature of this particular lesion, I think it is justifiable to suggest
the possibility that we had here an unusual complication of
scurvy due to a hemorrhage into the nerve-sheath or along its
course. Especially striking is the close connection, in time,
of the appearance of the facial paralysis and the first definite
scorbutic symptom, the pain and tenderness in the left knee.

In reviewing these cases the usual symptomatology of
scurvy is readily recognized. The insidious onset, the progres-
sive weakness, the general indisposition to work were followed
in the first case by "soreness" of the gums; in the second by
pain in the knees and soreness of the gums; in the third by the
facial paralysis and pain and tenderness about the left knee.
The involvement of the gums, the hemorrhages into the deeper
tissues, the purpuric spots, the pain about the joints and the
swelling thereof, the progressive weakness and dyspnea on
exertion, all of which symptoms have been found in these cases,
are classical phenomena, which may be regarded as diagnostic.
Two of these 3 men had the typical mental apathy on which
so many writers have laid stress—a "let-us-alone" attitude,
without interest in their surroundings or inclination to respond
to questions. In the case before you there is a history of hema-
turia; in the other two there were neither visceral nor mucous
membrane hemorrhages. These, in fact, are not cardinal symp-
toms of scurvy, though they occur, especially epistaxis and
hemorrhage from the bowel. You will recall that Sydenham
emphasized the tendency to nosebleed. Some authors have
noted blood in the urine as the single presenting symptom.
Some authors have spoken of a ravenous appetite; the patient
here says his appetite is poor; one of the other two had a desire
for food, but could not eat on account of his sore mouth. Of
the 3 patients, one complained of constipation, another said
his bowels were regular; in the third the condition was not

noted. The gastro-intestinal symptoms of the disease are variable.

Except the two records of slight fever on the chart of the patient here, fever was not present in any of these cases. In the absence of complications fever is uncommon, a point not to be overlooked in the consideration of infection as the etiologic factor. No murmurs were heard in any of our cases, though so-called hemic murmurs occur. In looking through the charts of the 18 cases treated in the hospital last year I found that murmurs were recorded only twice, and in one of these cases the murmurs were not dependent on the scurvy. In the 3 cases under discussion the pulse-rate was never over 100.

Two of the 3 cases have shown a marked secondary anemia. While moderate anemia is usual in scurvy, severer grades are not common, at least, in the comparatively mild type of the disease which we usually see here. There were complete blood examinations made on 10 of the 18 cases treated here last year; only 3 of these showed the erythrocytes below 4,000,000. One of these showed an especially interesting feature; the initial count showed erythrocytes, 2,110,000; seven days later the count was 1,649,000, though a note was made that the patient felt better and was improving; twelve days later the count was 4,000,000. Experimental work has brought out the fact that the disease is not likely to show amelioration of the symptoms for several days after treatment is begun; some time is required to obtain the vitamin effect. On the other hand, the vitamin effect lasts for several days after the antiscorbutic diet is withdrawn. There is usually no increase of the leukocytes; one of our cases had 11,750. Of the 18 cases admitted last year, only 2 had leukocytic counts about 10,000; the highest count was 12,600. Bierich, who did Red Cross work in Russia, reported his observations on 1343 cases; some of the symptoms he noted may be of interest to you. The ribs were tender to pressure in some cases, even causing an interference with deep breathing; separation of the cartilages was occasionally seen. Hemorrhagic pleuritis was noted 63 times, ascites 16, and pericarditis 4. Retinal hemorrhage was found 72 times, and

central nervous hemorrhage was diagnosed 18 times. He makes the statement that "rather frequently polyneuritides appeared to be produced by bleeding about the sheath of the nerve," a very interesting statement to us because of the facial paralysis already described, but he does not say that he saw peripheral nerve paralyses. Infarct of the lung was seen 6 times. He also states that sometimes hemorrhages were found at the time of an operation because fresh wounds bled easily and showed soft granulations. Major Turner, R. A. M. C., with the British Army in Mesopotamia, reported in 1917 that scurvy had not been frequent, but three groups of cases presenting a sort of latent scurvy were described: (1) those in which scurvy is the explanation of some otherwise obscure hemorrhage; (2) those in which it explains the sluggish healing of some wound or ulcer; (3) those that occur in the course of some other illness for which the patient has been fed on sterilized foods for long periods.

The myoidema noted in one case was probably without significance, although in 2 of these cases the reflexes were unusually brisk, and Darling has pointed out the presence of exaggerated reflexes, tachycardia, and dilatation of the heart in scurvy, and has called attention to certain resemblances between scurvy and beriberi. Hyperesthesia of the extremities is not uncommon; the myoidema was probably due to unusual muscular irritability. Night-blindness has often been described; it is probably the result of exhaustion rather than a symptom, essentially scorbutic.

Scurvy, infantile or adult, is one disease. The essential pathology of the disease is the same; infantile scurvy manifests itself especially in lesions of the developing bones; adult scurvy is not likely to show any great involvement of the bones. Infantile scurvy manifests almost no signs of disease of the gums; adult scurvy has this symptom as an outstanding feature. The gums prior to the eruption of the teeth are almost entirely free from involvement in scurvy. In general, the pathology of scurvy is that associated with the tendency to hemorrhage, and with perversion of development or regressive changes in

the bones. The hemorrhagic manifestations of adult scurvy have been described under the symptomatology. The scurvy of infants is characterized by subperiosteal hemorrhages and by hemorrhages within the marrow; there is rarefaction of the bones, sometimes with fractures, often with separation at the epiphyseal line.

In neglected cases the deep hemorrhages may result in deep ulcerations. Hemorrhagic exudates into the serous membranes occur; LeCount has noted the fact that hemorrhages into the leptomeninges may occur to such an extent in some cases as to seem to be the cause of death. The gastro-intestinal mucous membrane may show congestion, erosion, or ulceration. There is often fatty degeneration of the parenchymatous organs. The gums are edematous, hemorrhagic, ulcerated; the teeth loosen and may fall out; there may be necrosis of the alveolar · processes. Ulceration of the tongue and diffuse stomatitis may occur. Gangrene of the lung has been noted.

Before leaving the subject of the pathologic changes we want to quote from a suggestive editorial which appeared in the *Lancet* reviewing certain work of Zilva and Wells. The editorial is a discussion of certain aspects of experimental scurvy, especially as to the relationship of scurvy to disease of the teeth; thus, "The results have led to the conviction that the mildest degree of scurvy, which could just be discovered with the naked eye at the postmortem examination, produced well-defined microscopic changes in the structure of the teeth, and in the numerous examinations made the authors failed to observe a single exception to this statement; . . . the authors hesitate to draw definite conclusions from their work at this stage, but, as they point out, no satisfactory explanation has yet been advanced for the great prevalence of dental decay among civilized communities. Their investigation certainly suggests that deficiency in diet may well form a reasonable working hypothesis on which to base future research."

The diagnosis of adult scurvy rests on: (1) the inflammation of the gums, (2) the presence of the characteristic hemorrhages, deep and superficial, (3) the constitutional symptoms,

languor, apathy, dyspnea, (4) a more or less severe grade of anemia without leukocytosis, (5) the history of a poorly balanced diet, (6) the prompt response to treatment. Where the disease is well developed, to think of it is to make the correct diagnosis. The observations of Turner and Bierich are valuable in suggesting the importance of keeping in mind the symptoms of latent scurvy.

Differentiation must be made from diseases of the hemorrhagic group, and from severe forms of stomatitis. Acute leukemia will be recognized from the blood-picture. Purpura hæmorrhagica is characterized by a greater tendency to hemorrhage from the mucous membranes and the absence of the gingivitis. Purpura rheumatica is devoid of the gingivitis and of the deep hemorrhages; there are joint swellings associated with purpuric spots and symptoms of constitutional disturbance, fever, etc., of variable degree. The diagnosis of symptomatic purpura, infectious and toxic, is usually easy; these types show neither deep intramuscular hemorrhages nor gingivitis. Mercurial stomatitis may be confused with scurvy, but not easily, if the hemorrhagic tendency of scurvy is borne in mind. Vincent's angina has a febrile course and does not show hemorrhages; while the gums are often involved, the disease is essentially a stomatitis.

It was to be expected that the appearance of a disease with such distinctive symptomatology and such a capability of working havoc on a large scale as scurvy would cause much speculation as to its origin and nature. For three centuries or more these theories regarding the etiology and pathogenesis of scurvy have been susceptible of classification into two main groups, one explaining the disease as infectious, the other as nutritional. The word "infectious" is used in a broad sense to include all the older theories, which assumed the disease to be the result of miasmatic influences or of contagion; it is obviously absurd to apply the word "infectious" in the narrower sense to theories antedating the era of modern medicine. In the last two decades of the last century various observers believed that they had discovered a specific organism causing the disease. Recently

Jackson and Moore and Jackson and Moody have reported results of bacteriologic studies in experimental scurvy, which may be interpreted as suggesting an infectious origin of scurvy. In general, the consensus of opinion is against infection as the essential cause of scurvy. Moore gave his own opinion thus in 1919: "Further experiments are necessary to determine the significance of the presence of bacteria in scorbutic lesions. It is highly probable that some lowering of the resistance due to the one-sided diet permits the invasion of bacteria. Personally, I have considered them secondary invaders, gaining entrance after some break-down of the natural body defences. If, as some investigators assume, there is increased permeability of the intestinal mucous membrane, we must still be given some plausible explanation for this change."

Of late years much work has been done on the nutritional aspects of scurvy. Theobald Smith in 1895 noted a hemorrhagic disease occurring in guinea-pigs fed on cereals without succulent vegetables. However, it is since the work of Holst and Fröhlich in 1912 that the study of experimental scurvy has been greatly advanced. In 1914 Funk published his work on vitamins, describing rickets, pellagra, beriberi, and scurvy as "avitamoses." McCollum, whose theory of scurvy we will discuss later, maintains that thus far only beriberi and a peculiar eye affection in which the tissues surrounding the orbit swell until the eyes are not to be opened, and which is associated with an inflammation of the cornea, are the only two syndromes correctly included under the term "deficiency disease." The latter of the two diseases mentioned is thought to be due to the absence from the diet of the vitamin known as fat-soluble A.

The tendency of these investigations carried on so actively for the last few years has been to give a decided impetus to the opinion that scurvy is the result of an absence from the dietary of an accessory food factor; whatever the nature of this substance may be, we know it exists most abundantly in fresh vegetables and raw fruits, particularly cabbages, tomatoes, lemons, and oranges.

In July, 1919 a report containing in brief the essential practi-

cal results of the study of the food deficiency diseases was issued by the British Committee on Accessory Food Factors. The accessory food factors are recognized as three in number:

1. Antineuritic or antiberiberi factor, identified with the "water-soluble B" growth factor of the American investigators.

2. "Fat-soluble A" growth factor or antirachitic factor.

3. Antiscorbutic factor.

To quote further: "As far as is known the accessory food factors cannot be produced by the animal organism, and all animals are dependent for their supply directly or indirectly upon the plant kingdom." In regard to the antiscorbutic factor the Committee says, "This vitamin is necessary in a diet for the prevention of scurvy and is found in fresh vegetable tissues and (to a much less extent) in fresh animal tissues. Its richest sources are such vegetables as cabbages, Swedes, turnips, lettuces, watercress, and such fruits as lemons, oranges, raspberries, and tomatoes. Inferior in value are potatoes, carrots, French beans, scarlet runners, beet roots, mangolds; and also (contrary to popular opinion) lime juice. Potatoes, although classed among the less valuable vegetables as regards antiscorbutic value, are probably responsible for the prevention of scurvy in northern countries during the winter owing to the large quantities which are regularly consumed. Milk and meat possess a definite but low antiscorbutic value. The vitamin suffers destruction when the fresh food-stuffs containing it are subjected to heat, drying, or other methods of preservation. All dry food-stuffs are deficient in antiscorbutic properties; such are cereals, pulses, dried vegetables, and dried milk. Tinned vegetables and tinned meat are also deficient in antiscorbutic principle. In the case of tinned fruits the acidity of the fruit increases the stability of the vitamin and prevents to some extent the destruction which would otherwise occur during the sterilization by heat and the subsequent storage."

An interesting sidelight on the Committee's statement regarding the low antiscorbutic value of meat is found in an article by Stefansson, the Arctic explorer, which he contributed to the Jour. Amer. Med. Assoc. in 1918. He discussed a few

cases of scurvy which occurred at Melville Island in the winter of 1916–17, and states that they were promptly cured by the free use of meat, two-thirds of the daily ration being eaten raw, much of it frozen, a goodly portion of it tainted. Stefansson believes that, left to himself, the ordinary man will eat enough fresh meat to prevent scurvy; cooked fresh meat is probably a prophylactic against scurvy, though excessive cooking or keeping the meat after cooking lessens its antiscorbutic value; once scurvy is present, if meat is to have a curative effect, it must be eaten rare or raw. A recent article in the *National Geographical Magazine* has some interesting contents in this connection; it is a description of the Mongol people from the pen of Lord Bryce. He says that a Mongol eats no bread and rarely eats vegetables. His chief item of diet is boiled meat, the soup of which he drinks; the daily ration is said to amount to some 5 or 10 pounds of mutton or beef. The article is accompanied by no pictures of a scurvy-stricken people, nor does the text give stories of the prevalence of this disease.

Though the real nature of the antiscorbutic substance is not known, many of its qualities may be spoken of with considerable assurance. Many of the influences to which it is most sensitive have been mentioned; more details are constantly being added. Harden and Zilva found that the addition of bicarbonate of soda or any alkaline substance to green vegetables during cooking hastens the disappearance of the antiscorbutic factor. This factor, weakened, is often retained after cooking, but will disappear entirely if the cooked food is allowed to stand. Drying weakens or destroys the antiscorbutic factor; this appears to vary with the rapidity with which the drying is carried out and the degree of heat to which the substance is exposed. Different vegetables react differently to drying. The antiscorbutic value of cow's milk is likely to vary with the time of year, and especially with the availability of pasture and green foods. Germinating peas, beans, and lentils have a greater antiscorbutic value than the dry; this has been shown by Chick and Delf, who refer to the work of Major Wiltshire for practical confirmation of their experimental

work. Wiltshire while in Serbia used beans soaked for one day in water, allowed to germinate for forty-eight hours at room temperature, and cooked for only ten minutes, which served to make them palatable. Two groups of patients of equal number were under treatment; to one group lemon juice was given as the therapeutic procedure, to the other germinating beans; the results were almost alike; the beans, used thus, appeared to have fully as much antiscorbutic value as lemon juice.

McCollum does not accept the "food deficiency" theory of scurvy as proven, and holds that the advocates of the theory may serve it best by explaining certain facts not in harmony with the theory, rather than by heaping up new facts to prove this "attractive hypothesis." McCollum and Pitz, on the basis of their experimental work, believe that constipation is an important factor in guinea-pig scurvy. They express the opinion that, on a diet of succulent vegetables, bulky feces are formed which are readily eliminated; but on a diet of oats and milk the feces are pasty, there occurs an impaction of feces, and there may be sufficient injury of the intestinal wall to permit the entrance of bacteria or the absorption of toxins. They refer to the work of Jackson and Moody, and find in the work of these observers support for their own opinion that "the undue retention of feces is the primary cause of the development of experimental scurvy in the guinea-pig. Secondarily, toxic products of bacterial origin or the invasion of the body by bacteria are causal factors."

The idea that constipation is the effective cause of scurvy has not found much support among the workers in experimental scurvy. Hess, Cohen and Mendel, Cohen and Givens, and the English workers, Harden and Zilva, Chick, Hume, and Skelton, are advocates of the "food deficiency" theory.

Various other theories of the disease have found favor from time to time; since the theory of a deficiency of potassium found such wide acceptance three or four decades ago, many ingenious theories attributing the disease to various manifestations of perversion of the mineral metabolism have been proposed.

The relationship of phosphorus and calcium have both been subjects of study in recent years, thus far without result. Gerstenberger suggests a perversion of carbohydrate metabolism due to the absence from the dietary of some substance necessary to the furtherance of normal carbohydrate metabolism. He quotes from Braddon and Cooper and from Funk the opinion that a certain amount of vitamin is necessary in proportion to the amount of carbohydrate taken, and refers to a case of his own, a child thirteen months old, who had for some time prior to his being brought under Gerstenberger's care nothing but raw milk diluted with water and an enormous amount of Mellin's Food. Stefansson is inclined to take the medical profession to task for its failure to investigate more thoroughly the effect of common salt in producing scurvy; salted meat has been blamed for a long time, but Stefansson sees the cause in the salt, though the meat kept in salt does lose its antiscorbutic value.

Mental factors probably play a part in the production of the disease. A monotonous life, worry, anxiety, and depression predispose to the disease. Major Turner, writing from Mesapotamia, and Bierich, from Russia, express independently the opinion that psychic (depressive) factors are of great moment.

This discussion of etiology leaves little to be said about treatment. The prophylaxis is obvious; perhaps the old tradition of the necessity of a "spring tonic" is based on fundamental deficiencies in the diet usually taken in the winter months. By the time spring comes we may all be nearer scurvy than has been appreciated. At all events it must be emphasized that a well-balanced dietary is of the utmost importance, and the problem of the preservation of foods is not entirely solved so long as the vitamins are not preserved.

Fruit juices and fresh vegetables are indispensable articles of a safe dietary. The curative treatment employed in this hospital is the administration of ward diet plus fresh fruits three times a day, and salads twice a day; lemon juice is also given freely. Iron is usually given for the anemia. The mouth is cared for by a proper mouth-wash, and astringent applica-

tions to the gums. It might be well to note that our patient had eaten bananas freely, but no other fruit. Bananas are particularly low in antiscorbutic quality.

Harden, Zilva, and Still have used a concentrated lemon juice; they were able in this way to give up to seven times the amount which can usually be given daily. The results were striking, improvement being very rapid. Not the least interesting feature of this report is the fact that the results in the human infant coincided with the results obtained in the experimental scurvy of guinea-pigs. Hess has used orange juice, boiled and rendered alkaline, intravenously, in doses of 1 ounce, with excellent results; Hess also has called attention to the value of dried orange peel as an antiscorbutic; this substance will retain antiscorbutic properties for three months or more.

As to prognosis, any extensive discussion would be superfluous. Here, at the County Hospital, the mortality is low; most of the patients leave in good condition within a month after admission. When the conditions are favorable for treatment the desired response is almost invariable; the prognosis depends almost entirely on the possibility of providing the patient with proper food before his condition becomes too serious.

Note.—January 27, 1921. The patient with scurvy whom you saw two weeks ago is ready to leave the hospital. His legs are still a little stiff, the gums are very much better; he is eating well, and though a little weak, wants to go home. You will recall that this patient had a well-marked secondary anemia. On admission his blood examination showed hemoglobin 34, erythrocytes 3,000,000. leukocytes 11,750; on January 17th, shortly after you saw him, the results were: hemoglobin 32, erythrocytes 2,410,000, leukocytes 15,100. At the same time it was noted that the "mouth is much better and improvement continues." The examination today shows hemoglobin 63 per cent., erythrocytes 4,090,000, leukocytes 4650.

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FOUR NASAL CASES AND AN AORTIC ANEURYSM WITH LARYNGEAL SYMPTOMS

TODAY we shall be able to show you cases illustrating several nasal and one laryngeal condition, some of them rather common, others perhaps less frequently seen. Our clinic today is a sort of review, and an inspection of cases which have been under observation by us for varying degrees of time. It is important, whenever possible to do so, to see the end-result of any particular line of treatment. Only then is it possible to arrive at some degree of judgment regarding the efficacy of one or the other therapeutic measures.

The first 2 cases to be presented show considerable similarity in symptoms referable to the nose.

Case I.—Miss I. C., age twenty-one, stenographer, came to us a few weeks ago just as she was recovering from a severe attack of tonsillitis and nasopharyngitis with much postnasal "dropping." There was a history of very frequent attacks of rhinitis, with marked paroxysms of sneezing and a profuse watery discharge from the nose. Intermittent nasal obstruction alternately from one to the other side has been present for · some time, causing some mouth breathing.

Examination of the nose showed some deviatio septi, a boggy left middle turbinate, and intumescent inferior turbinates. In the nasopharynx there was a small adenoid remnant. The faucial tonsils were submerged, but contained no pus. Trans-illumination of the nasal accessory sinuses was negative.

After cocainization of the middle meati a saturated solution of trichloracetic acid was applied, and a 1 per cent. silver nitrate solution used in the nasopharynx. The patient returned a week

later feeling somewhat relieved. At this time we infracted the middle turbinates away from the lateral nasal wall, thus better exposing the meati, and again applied the trichloracetic acid.

Today the patient states that the sneezing and watery discharge have practically ceased. We will once more apply the trichloracetic acid and then dismiss the young lady for a month's time. We always lengthen the interval between treatments as soon as any improvement is noted.

Case II.—Miss B. D., age twenty-one, clerk; presented herself some time ago with a history of considerable nasal obstruction, and frequent sneezing, accompanied by profuse watery discharge. There have been occasional attacks of sore throat, and during the past six months at times a "choking" sensation, due probably to some enlargement of the thyroid gland. Rather frequent epistaxis is said to occur, as well as vertex headaches, the latter usually at night. Tonsillectomy and adenoid removal were performed three years ago.

Examination some months ago showed as follows: In the nose considerable intumescence of the inferior turbinates together with some hypertrophy of their inferior edges; small septal crests; a negative nasopharynx. The pharynx showed absence of the tonsils. The thyroid gland, was moderately enlarged, but soft.

The patient was accustomed to using much face powder, and knowing this to be an irritant, and often an exciting factor in these nasal conditions, its use was interdicted. At the same time the inferior turbinates were touched with a bead of silver nitrate and an astringent was prescribed for use in the nose. The patient returned a week later feeling only slightly better, and confessed that she was still using the face powder. She then disappeared until a week ago, at which time after cocainization we infracted the middle turbinates away from the lateral nasal wall, and applied trichloracetic acid to the depths of the middle meati. Today you have heard the patient tell of the amelioration of her nasal symptoms, due perhaps in part to a change in the kind of face powder used, but largely, I believe, to the manipulation employed by us. We will repeat the

application of the trichloracetic acid this afternoon. We suspected that the headaches originally complained of were due to eyestrain. The patient had had glasses for some years, but rarely used them. We insisted that she wear them, and you have heard her admit that since doing this the headaches have practically disappeared.

It has been our experience especially during the past few years that the very extensive and indiscriminate use of perfume, face powders, and rouges has in many young women produced intense irritation, causing sneezing, nasal discharge, lacrimation, etc. Substituting a simple talcum or borated talcum powder has in many instances greatly if not entirely relieved the symptoms. There is a group of cases so often seen in clinic and practice, in which there is a history of very frequent so-called "head colds" (occurring sometimes as often as twice a week), with sneezing, profuse watery discharge, but no acute inflammatory conditions. Examination at that time may show an intumescence of the inferior turbinates, with a boggy, edematous appearance of the middle turbinates, and a swelling of the mucosa covering the middle meati.

The etiology of this so-called hyperesthetic rhinitis is not absolutely definite; but we know that inhalation of certain irritants like the above-mentioned face powder, or a protein sensitization in the case of certain foods, or a disturbance in basal metabolism, etc., are factors in the causation of these symptoms.

The pathology is often that of a hyperplastic ethmoiditis with thickening of the mucosa of the ethmoid cells, but without any pus.

In the line of treatment many things have been used, but in some cases nothing has availed. In some others, however, minor measures such as those employed in our cases have given much relief. We have observed some cases months and even years after treatment had been discontinued, who were still comfortable in every way. In the effort to relieve the patient we believe that the most conservative measures should be first tried. Most important is the determination of the etiologic

factor, and then eliminating irritants such as face powders or certain food proteins if the patient shows any idiosyncrasies. The use of thyroid gland is indicated if the basal metabolism is impaired and lowered. Injection or blocking of the nasal sensory nerves with alcohol (as practised years ago by Dr. O. J. Stein in hay-fever) may prove valuable in some cases. The use of trichloracetic acid is very good in a large percentage of cases, but in order to thoroughly reach the depths of the middle meati we usually infract the middle turbinates and press them away from the lateral nasal wall, thus widening the space leading to the meati. As a last resort opening of the ethmoid cells, together with removal of polyps, if any, is at times necessary.

An entirely different clinical picture is presented by our next case both from the standpoint of her history and that of therapy.

Case III.—Mrs. H. V., age fifty-eight, housewife; came to us about six months ago, and while she is now apparently entirely well, and simply returns periodically to report on her condition, it may interest you to know her history.

When we first saw her she stated that for some time previously she had had a marked purulent discharge from the right naris, unaccompanied by pain. One operation had been performed, but without any improvement of the symptoms, so the attending rhinologist had told her that a radical operation on the right antrum was indicated. On examination at that time we found the nasal septum rather markedly deviated to the right side, and pus in the right middle meatus. Transillumination showed a dark right maxillary antrum, and an x-ray picture also showed considerable cloudiness of the right antrum and frontal sinus, together with apparently some change in the alveolar process. All the teeth were gone and the patient was using an upper and a lower plate. The right antrum was irrigated by way of the natural opening in the middle meatus and a large amount of pus evacuated. It was only at the second or third treatment that more careful examination revealed what had been overlooked by the man who had previously treated the patient, and what I had also failed to note at the first exami-

nation, namely, a small fistula in the anterior portion of the hard palate just behind the alveolar process. At first we thought pressure of a badly fitting plate might have caused erosion and later necrosis, but a dental x-ray film revealed an unerupted right upper lateral incisor, with necrosis at its distal end. Enlarging the fistula slightly, the tooth was removed and the tissues cureted. The fistula on probing showed no direct connection with the antrum; but that the necrotic unerupted tooth was the main factor in causing and continuing the sinusitis was shown in the very rapid improvement after the extraction. A few more irrigations of the antrum apparently eliminated the infection. Today merely as a test we have washed the antrum of Highmore, and you have seen the irrigating fluid return clear. The fistula in the hard palate has entirely closed.

This case shows how a tooth, even an unerupted one, may cause severe maxillary antral infection, and also emphasizes again the importance of a thorough examination to determine the etiology of a sinusitis, especially that of the antrum of Highmore. Often the attention to details of investigation together with conservative local measures may bring about a speedy and complete recovery, and thus obviate the necessity of either prolonged, at times painful, treatment, or that of radical operative procedure.

We will now show you a case presenting abdominal and pelvic symptoms which stand in certain relation to, and are sometimes influenced by, manipulations of the nose.

Case IV.—Miss R. H., age sixteen, schoolgirl; about seven months ago this young lady first came to the clinic with a history of having had for one and a half years very severe dysmenorrhea following an appendectomy. Not only did she have severe pains in the abdomen and the back, but nausea followed very often by vomiting was also present. In fact, for several days of each menstruation the patient was completely incapacitated and usually confined to bed most of the period. Frequent head "colds" are said to occur and at times there is considerable nasal discharge of a mucous character. Tonsillectomy was performed seven years ago.

Examination of the nose showed intumescences of the inferior turbinates and some swelling of the middle turbinates and of the tubercula septi. Transillumination of the nasal accessory sinuses was negative. The pharynx showed a smooth normal mucosa; the tonsils were gone. The neck showed a few moderately enlarged lymph-glands.

The patient was told to return at the time of the next menstruation as soon as the severe cramps might appear. However, at the next period she was so sick because of pain, vomiting, etc., that I went to her home. Without any suggestion or hint as to the procedure to be used, I applied a 10 per cent. cocain solution to the anterior third of the inferior turbinates, to the tubercula septi (which as you know are the thickened areas at the junction of the middle and upper thirds of the nasal septum), and to the edge of the middle turbinates. Within a few moments the severe pain subsided, the vomiting failed to recur, and the patient was very comfortable. She was instructed to return to us ten days later, at which time, and again a week later, after cocainization, trichloracetic acid was applied to the septal tubercula, the edge of the middle turbinates, and the anterior third of the middle turbinate.

The patient reported to us that the succeeding menstruation was painless and free from nausea and vomiting. Likewise were the two following menstrual periods, but at the fourth one some abdominal pains appeared which were at once relieved by cocainization. Trichloracetic acid was applied during the quiescent time, as previously mentioned. The last two menstruations have been painless, and we will today carefully apply the galvanocautery to the tuberculous septi and the anterior ends of the inferior turbinates in the endeavor to give permanent or at least long-standing relief to the patient.

The so-called "genital areas" in the nose, namely, the inferior turbinates and the tubercula septi, usually show certain changes at the time of menstruation. They swell (but according to J. N. Mackenzie there is congestion of the entire nasal mucosa during menstruation), they bleed easily, they are painfully sensitive to palpation with a probe, and they are somewhat

cyanotic. There are two groups of dysmenorrhea—one in which the pains disappear when the flow begins, and the other in which the pains continue after that time. These pains have two localizations: in the lower part of the back and in the abdomen. It has been shown that cocainization of the tubercula septi usually relieves only the lumbar pain, and application to the inferior turbinates suspends the abdominal cramps. A further highly interesting and important point is the fact that cocainization of one side of the nose (c. g., the right) influences mainly the pains in the opposite (c. g., left) side of the body, thus indicating a crossing in the sensory paths similar to that of the optic nerve.

We have had excellent results from this line of treatment in a fair number of severe dysmenorrhea cases. In many quarters great skepticism has been expressed regarding the relief of menstrual pains by cocainization, the objectors claiming that the effect, if any, was due to suggestion. However, this factor can easily be eliminated by not informing the patient beforehand that anything special is to be done. Nevertheless, the relief is often almost miraculous in its speed and extent. Furthermore, the duration of relief following cauterization with the trichloracetic acid or the galvanic current certainly points to definite therapeutic action and not to suggestion. Fliess it was who first called attention to this method of relieving dysmenorrhea; and in this country Emil Mayer among others has done much work along this line. The exact anatomic explanation for the phenomenon has not been entirely clear, but certain segments of the spinal cord supplying the pelvic region are connected by way of the sympathetic nervous system with the areas in the nose which we have mentioned, and by influencing the latter we produce effects in the regions supplied by the former. Of course, it is *not* to be assumed that all cases of menstrual pain are amenable to nasal manipulations. Dysmenorrhea may be caused by mechanical (affections of the uterus, etc.) and other factors, and it is the gynecologist who will have to determine in each case the probable cause of the pain. The attempt, however, to relieve the cramps by cocain-

acute laryngitis causes impairment of the voice for a short time only) may be due to a serious lesion in the larynx. Often this symptom is neglected by the family physician and no inspection of the larynx is made, or, if so, it is done when the condition has advanced very far. In most cases of persistent hoarseness, tuberculosis, malignant disease or syphilis of the larynx, or the paralysis of a cord is to be found. In order to be of avail early institution of treatment is most important, and in order to do this an early diagnosis by means of the most careful examination of the larynx is imperative.

CLINIC OF DR. ARTHUR F. BYFIELD

COOK COUNTY HOSPITAL

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AIDS IN PHYSICAL DIAGNOSIS

I HAVE planned to use this period with an interpretative discussion of certain matters of physical diagnosis rather than with the usual presentation of clinical material. As the time at my disposal limits me to the selection of a very few subjects in this field and to a brief treatment of these few subjects, I have chosen several—largely unrelated—points in technic which have rendered me good service in this phase of diagnosis, and which will, I hope, help you over certain difficult spots in your clinical work. I might add that the methods I advise and the opinions I put forth do not in some particulars meet with the generally accepted views on the several subjects; to this I can answer simply that my personal experience has found them reliable and of service.

A. **Pleuropulmonary Diagnosis.**—1. *The Relative Weight of the Physical and the General Clinical Findings in the Diagnosis of Early Pulmonary Tuberculosis.*—You will allow me first of all to touch upon a general theme, a very old theme, but one which almost daily confronts every one of us. and which will bear all the emphasis we can give it. I cannot agree with those who presume to base a diagnosis of incipient pulmonary tuberculosis purely on a slight change in the percussion note over an apex or other relatively small pulmonary area: on a variation from the normal vesicular breath sounds: or even on the presence of a few fine râles. Given one or even all of these anomalies in the face of a negative symptomatic picture of tuberculosis—temperature. pulse-rate. weight, appetite. blood-picture. etc.—it is surely a hazardous thing to render the verdict

tuberculosis, *unless, of course, bacilli can be demonstrated in the sputum.* You will find that the diagnosis of pulmonary tuberculosis is easily enough made, but only with the greatest difficulty unmade in later months or years.

Lesions long since healed may leave evidence behind them in the shape of a small area of impaired resonance or in the form of circumscribed harsh breath sounds. Atelectatic areas, such as are found not uncommonly in cardiac lesions, in emphysema, in thoracic deformities, and in shallow breathers may give all the usual signs of infiltration even over an apex; while râles of fairly constant character are common over the apices in individuals who have never learned to ventilate their lungs completely, in patients with nasal obstruction, and in not a few for no discoverable reason. Osler has put the point under discussion very happily, as follows: "Practically, in these early cases we have two groups—the one with symptoms and no physical signs, and the other with physical signs and no symptoms. Of the two, the former is of the greater importance."

To conclude the matter, we might sum up things in this way: A positive diagnosis of pulmonary tuberculosis is unwarranted and unfair when based solely upon one or several slight variations from the normal in physical findings. These— particularly the shortened note and the roughened breathing —prove not so infrequently, as the case is studied by other methods, either to have had little basis in fact or to be susceptible of other interpretation. Only the fully rounded-out clinical picture, associated with unmistakable pulmonary findings and reinforced by the laboratory, warrants a diagnosis fraught with so great a significance to the patient.

2. *A Method of Provoking Râles Over a Suspected Area.*— Some years ago I called attention to a method I had frequently found valuable in bringing out râles in a suspected area—a method which I have been informed has been of service in the hands of others. A sharp cough at the end of expiration is the common procedure to produce moisture; if this proves unsuccessful, then the aid I am about to mention is in order. It consists in having the patient count from 1 to 10—or 1, 2, 3

a number of times—in a whispered voice, without taking a breath until the end of the count is reached, when a long inspiration is made. By this simple procedure I have many times noted râles over a suspected area, after cough has failed to produce them. Indeed, I have felt reasonably secure in ruling out moisture over an apex at a particular examination if the whispered count fails to elicit râles.

Fig. 39.—Percussion of the hepatopulmonary line in a woman with a moderately large breast. The points represent the course of percussion from the midclavicular to the midaxillary lines. The upright position of the patient gives one only a fair idea of the extent to which the breast interferes with percussion.

I shall make no more of an attempt now than when I originally published the details of this method to explain the basis upon which it rests.

3. *Pulmonary Percussion in Women with Large Breasts.*—Accurate determination of the pulmonary bases anteriorly in women with large breasts is often out of the question. An exact determination is more likely to be desired on the right than on the left side, and from the pulmonary as well as from the hepatic standpoint. This difficulty can very easily be met

by shifting the percussion axis from the usual midclavicular plane toward the anterior or midaxillary regions as one approaches the third to the fourth rib (or interspace), depending upon the size of the particular mammary gland (Fig. 39). It is necessary merely to bear in mind the varying level of the hepatopulmonary boundaries at the several lines (upper edge of the sixth rib in the parasternal and midclavicular regions, eighth to ninth ribs in the axillary line, the border therefore following very nearly a horizontal line).

4. *The Grocco Sign.*—This is the contralateral paravertebral triangle of dulness described originally as part of the percussion findings in pleural collection of fluid. The majority of writers who touch upon the matter criticize it adversely and harshly. It has been urged, for example, that it is present in too many conditions other than pleural effusion to be at all characteristic of the latter; and that the examiner is usually able to demonstrate the contralateral dulness only after he has satisfied himself that physical findings suggestive of fluid are present on the other side.

I disagree flatly with these criticisms, and must maintain that I have found the sign of considerable, often deciding, value in the distinction between pulmonary consolidation, pleural adhesions, and pleural fluid.

As in the case of any other isolated finding, the Grocco phenomenon must be allowed its weight only after proper interpretation. We all know that conditions causing upward pressure upon the diaphragm—fluid, tympanites, tumor, pregnancy—may in some cases produce the triangular dulness under discussion; we know that large pericardial effusions may do the same; we know that in cases of bilateral (basal) pulmonary pathology the Grocco dulness is concealed, and we know that in some cases of massive pneumonia the great increase in the size of the affected lung may produce pressure on the opposite side and the Grocco triangle.

Granting all this, I feel justified in recommending that you become familiar with the Grocco sign and that you allow yourself to place diagnostic weight upon it when the question is

whether in a case of pleural or pulmonary disease producing unilateral physical findings, the possibility of fluid must be considered, either associated with pulmonary consolidation, or as the sole abnormality.

5. The point to which I wish next to call your attention is also one of percussion, and applicable to the demarcation of the upper limits of dulness or flatness posteriorly. It is mentioned by a few writers, as, for example, Strümpell in his text-book. Ordinarily, of course, we percuss from above downward, passing from the zone of normal resonance—if any such be present on the side under examination—sharply or gradually, as the case may be, to the zone of unmistakable dulness or flatness. The determination of this boundary is frequently not easy, and as its exact location may be desirable both from the standpoint of the extent of the lesion and of the diagnostic character of the upper line—horizontal, corresponding to the interlobar fissure, oblique (Ellis, Garland)—any refinement in the method of determination is distinctly of value.

Such a refinement—if I may call it that—is often made possible by percussing from below upward instead of from above downward. Apparently the transition from flatness or dulness to the normal or the relatively normal is better appreciated than is the converse.

6. Two procedures which, in view of what they teach us, are often neglected in the routine examination of the chest are an examination to determine the presence of the Litten sign and the degree (on percussion) of respiratory mobility of the pulmonary bases. My experience has taught me to regard a clear-cut Litten phenomenon, if reinforced by a sharply marked descent of percussible pulmonary resonance on full inspiration, as highly indicative not only of normal pulmonary tissue at the base—few pulmonary conditions are unassociated with a reactive pleurisy—but also of normal apices. It has never been entirely satisfactorily explained why apical disease should affect the signs under discussion; yet I believe that one is fairly safe in ruling out apical pathology if nothing points to pleural involvement at the base.

These are the few matters in pleuropulmonary diagnosis which I have selected for the present discussion. At another time, possibly, I shall return to this topic and emphasize some other points of every-day interest; now, however, I must pass on to a consideration of other organs in a similar fragmentary way.

B. **The Heart.**—1. *The Apex-beat.*—Next to auscultation, the determination of the location and character of the apex-beat gives us more information in the *routine* examination of a heart than does any other phase of physical diagnosis. In this connection I am considering the point of maximum cardiac thrust which clinically we have become accustomed to call the apex-beat, not the true apex, as determined by fluoroscopy, the latter being actually 2 to 5 cm. below the former. For practical and comparative purposes, however, we may still rely upon the maximal impulse.

In many cases the apex cannot be felt with the patient in the recumbent position; in perhaps only a small minority of cases, however (patients with muscular or fatty walls, emphysematous patients), does one fail to find the apex either by having the patient turn toward the left or having him sit up and lean forward.

The position of the apex thus found is at least a rough index of the size of the heart. In many cases it does not correspond to the actual left border of the heart, as determined by orthodiascopy, yet it probably furnishes more accurate information as to this border than does percussion at the hands of the average man. In a sufficiently accurate way, also, the position of the apex-beat gives us information as to the actual pathology present. Thus, an apex dislocated to the left and downward speaks for aortic disease (regurgitation, atheroma, aneurysm, with or without other cardiac involvement); conversely, a dislocated apex in the fifth interspace generally points toward the absence of these aortic processes; a fourth interspace apex-beat (in an adult) suggests pressure from below the diaphragm (fluid, gas, etc.); an apex dislocated to the right generally means left pleural effusion; less often, pleural retraction on the right side.

I make no apology for touching upon these elementary topics; it is far better to know the a b c's thoroughly and to be able to use them for all they are worth than to glide over the commonplace and attempt to rely upon the ever-increasing armamentarium of refined diagnostic methods.

Less familiar matters are the qualities of the apex-beat— its breadth, intensity, and definition. We palpate the beat between the thumb and index-finger. The normal beat is about 1.5 cm. in breadth (in the female a little narrower); its borders can be clearly felt, that is, the beat is well circumscribed, and can readily be marked with the skin pencil. This sharp delimitation is due to the fact that the apex-beat is normally formed almost entirely by the left ventricle. The normal apex may easily be caused to disappear by digital compression.

A broadened beat, especially if also intensified, speaks for ventricular dilatation and hypertrophy. The character of the broadened apex-beat, whether well or poorly defined or circumscribed, i. c., with boundaries readily "picked up" between thumb and index-finger, generally gives us considerable information as to what relative share the ventricles have in the formation of the beat. In a general way we may say that dilatation and hypertrophy of the left ventricle alone give a broad, well-circumscribed apex; that enlargement of the right heart alone causes an abnormally broad beat not easily picked up between the fingers, and that enlargement of both chambers produces a wide, diffuse apex-beat.

That right ventricular hypertrophy does not cause as marked a broadening of the apex as does a similar and equal condition of the left is explained anatomically by the fact that the muscle bundles of the left chamber are larger and thicker than those of the right. And right ventricular enlargement does not produce as circumscribed an apex-beat as does left because even under pathologic conditions the left ventricle forms most of the beat.

The position, breadth, definition, and intensity of the apex-beat may give us valuable information, therefore, not only as to the size of the left heart but also as to the part played by the

two ventricles in a cardiac enlargement; and frequently, according to the rules just laid down, we may obtain a very fair idea as to the actual clinical condition present, whether cardiac or extracardiac.

2. *An Aid in the Percussion of the Left Ventricular Border in Women with Large Breasts.*—This is often a matter of extreme difficulty which is not entirely obviated by having an assistant retract the gland. The difficulty is enhanced by the fact that it is often true in these cases that the apex-beat cannot be palpated.

The method I am about to describe is a rough one and cannot be depended upon so much to give the exact location—so far as percussion ever permits—of the left ventricular border, as to enable one to determine whether or not the left border is out, and, in a broad way, how much.

You are all familiar with the normal course of the left vessel and heart boundary line. If now, in our percussion of deep dulness from left to right we find the changes in note sought at the normal points in the second, third, and fourth interspaces (we assume that percussion in the lower interspaces is difficult because of the large breast), then we have a right to construct the remainder of the left border along normal lines. If, however, our points in the upper interspaces are displaced to the left, then—we emphasize again the fact that our conclusion is general and broad—we may assume that the remainder of the left border is proportionately displaced (Fig. 40). The method fails, as we have noted, in giving us information as to the special character—mitral, aortic, etc.—of the line of left cardiac enlargement.

3. *The Right Heart Border.*—I have spoken of the information regarding enlargement of the right heart that may be derived from a study of the apex-beat. Such information, however, can hardly be regarded as fully adequate to establish the diagnosis of dilatation and hypertrophy of the right ventricle.

Percussion of the right border is notoriously unsatisfactory because of the associated vibrations of the sternum itself. In my experience this applies rather to the normal right border

than to one associated with a large right heart. As a rule, dulness over the lower sternum, especially if in contrast to the usual and characteristic note of the upper sternum, speaks— in purely cardiac cases—for an enlarged right heart.

. In marked right ventricular hypertrophy we often find, in addition to lower sternal dulness, dulness of .variable degree to the right of the sternum, a heaving of the lower sternum, and a pulsation in the epigastrium.

Fig. 40.—The dotted line represents the construction of that portion of the left cardiac border percussion of which is rendered difficult by the large breast. The comment made under Fig. 39 relative to the patient's upright position applies equally well in this case. The enlargement to the left is here very moderate.

In some cases one can find no physical signs pointing definitely to right heart enlargement; then a knowledge of the cardiac pathology associated with the particular condition present may warrant one in assuming such an enlargement (for example, in pulmonary emphysema and in thoracic deformities).

4. I find that the average examiner in his routine study of a case does not palpate in the jugulum. This ought never to be omitted because it gives us direct information as to the· aortic pulsation. In the normal case only a soft impact can

be felt; in aortic disease the height of the pulsation in jugulo, its intensity, a thrill or a sharp rise, and sudden collapse may give us very definite diagnostic information (dilatation of the aorta, aneurysm, aortic stenosis, aortic regurgitation, hypertension).

C. The Liver.—1. *Technic of Palpation.*—We have been generally taught, I believe, to palpate the liver facing the patient. While this method seems to be satisfactory as a general thing, I would recommend to your attention palpation with the examiner facing the patient's feet. I have found this method valuable in the following ways: (*a*) It renders palpation easier in the case of abdominal walls which do not relax well; (*b*) it enables one more readily to palpate minor degrees of enlargement; and (*c*) it facilitates the determination of the character of the surface and edge of the organ—points to which we shall refer later.

2. *An Aid in Differentiating an Enlarged Liver from a Displaced or Ptosed Liver.*—I shall make no effort to take up with you the many clinical situations in which it is of importance to determine whether the liver is actually enlarged or only displaced downward. Valuable information can, of course, be obtained by determining the upper margin of hepatic dulness; if this lies abnormally high, it is fairly safe to assume that the low-lying liver margin speaks also for enlargement. At times, however, there are difficulties in the way of establishing the upper limit of the dulness (fluid, consolidation, etc.). Under these circumstances the position of the left border of liver dulness gives valuable information.

Under normal conditions, as we percuss from left to right along the left costal arch, we encounter dulness of hepatic origin not further to the left than the left parasternal line. Dulness to the left of this line indicates that a liver edge felt in an abnormally low position below the costal margin is part of an enlarged organ.

3. *The Lower Edge and Surface of the Liver.*—As to the character of the surface of the liver, I wish to make only a single comment, to wit, it behooves the examiner to be most conserva-

tive in diagnosing elevations or irregularities unless these be of considerable size. It is a question whether the small nodosities such as are met with in the Laennec type of cirrhosis are under any circumstances susceptible of palpation, a question still further complicated by the fact that the adipose tissue of the abdominal wall imparts a very similar "granular" sensation.

The character of the liver edge often gives considerable diagnostic information. The normal margin is moderately rounded; that of the Laennec cirrhosis, whether atrophic or hypertrophic, is generally sharp; while the edge of the Hanot type of cirrhosis, of the simple fatty liver, of the liver of passive hyperemia, and of other enlargements in general is likely to be blunt. The diagnosis of cirrhosis is strengthened by the definite hardness of the organ.

Percussion is also of assistance in determining the character of the liver edge. When palpation shows the lower hepatic margin to coincide with the line of lower hepatic dulness, we may assume that this margin is blunt; on the contrary, when the palpable edge is lower than the percussed line, we generally find that the edge is sharp. In the latter case bowel subjacent to the thinned-out margin interferes with the determination of the actual lower limit of hepatic dulness.

D. The Spleen.—*Palpation and Percussion.*—My own observations have led me to believe that with the average examiner palpation represents the sum total of the routine of splenic physical diagnosis. In one way I can offer no serious objection to this, inasmuch as one treads a rather unsafe ground who does not base his ultimate opinion as to whether the spleen is enlarged or not on his ability to palpate the edge.

A word, first of all, as to the technic of palpating the spleen. In the average case (appreciable enlargement of the organ; fluid or gas in the abdominal cavity not of sufficient degree to make palpation entirely out of the question) I have found that I am able to feel the edge of the enlarged organ just as readily with the patient in the dorsal recumbent position as when he turns toward the right. In this particular a great deal depends

upon learning *how* to palpate a spleen, and this is a matter solely of experience. The intern is fortunate who begins his hospital work under the supervision of one who is patient enough to stand by and show him why he does not feel a spleen that is certainly enlarged.

In a few cases I have found splenic edges not otherwise palpable by the method described in connection with the liver, namely, by facing the patient's feet and hooking the fingers of the palpating hand under the costal arch.

Palpation, as I have already noted, is the final criterion of splenic enlargement. Percussion, however, generally gives one very accurate preliminary information as to whether he shall be able to feel the splenic edge. You will read in your texts generally that splenic percussion is of comparatively little value. In a relative way this is true, as contrasted, for example, with the value of pulmonary percussion; in an absolute way, in so far as percussion gives us information concerning the anterior and lower borders of the spleen, I would take exception to the statement of the texts.

Splenic percussion is made along the long axis of the organ, *i. e.*, diagonally downward and forward, with the patient on his right side and his left arm raised above his head. The anterior edge normally does not extend, as a rule, beyond the midaxillary line and never beyond the anterior axillary line. The dulness of the normal organ, furthermore, does not reach the costal margin—you will recall the extent of the spleen from the ninth to the eleventh ribs. Therefore, when percussion is feasible—no undue amount of gas or fluid in the abdomen; no pleural fluid—and when percussion shows splenic dulness to extend farther forward than the anterior axillary line and downward to or beyond the costal border, we are fairly safe in assuming splenic enlargement, and palpation will confirm the assumption.

The question of splenic enlargement as against splenic ptosis does not confront us so frequently as a similar problem in connection with the liver (see above). When it does, we must rely upon the upper margin of splenic dulness—normally

at the ninth rib—and the clinical findings generally to give us the desired answer.

I have allowed myself a longer discussion of these selected topics than I had originally intended. Attribute this, if you please, to a personal leaning toward methods which cannot be regarded as hobbies, because they have been of value in the hands of others and because they too regularly give accurate results as controlled by other means.

The topics discussed represent only a small portion of the material in this field that I should like to go over with you; the remainder must be postponed to a later period.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

POSTERIOR MEDIASTINAL PLEURISY [1]

DURING the past few months some of the medical cases admitted to the Medical Service, Ward 61 have presented findings and problems in diagnosis of more than ordinary interest. It is proposed to present the records of 2, more or less related, as the substance for this clinic.

CASE I

Female, age thirty-three, admitted January 17, 1919, and sent to the "Flu Ward" as influenza.

History on Admission.—Sickness began two weeks ago with chills, headache, and fever. Confined to the bed for a few days, when she got up and about for four days, but did not feel well. During past week has been confined to the bed with a. cough, which is non-productive and much worse at night. No bloody expectoration. Fever, nausea, and vomiting have been present during past week. Complains of some "chest pain" on breathing, but especially on coughing. which is present on either side.

Physical Findings.—Examination was negative except for lung findings, which consisted of diffuse râles over entire chest, anterior and posterior. Slightly impaired resonance, with bronchovesicular sounds over lower portion of both lower lobes posteriorly. Subsequently these signs over the lower lobes became more pronounced and the diagnosis of a flu-bronchopneumonia was made.

[1] Submitted May, 1920.

FREDERICK TICE
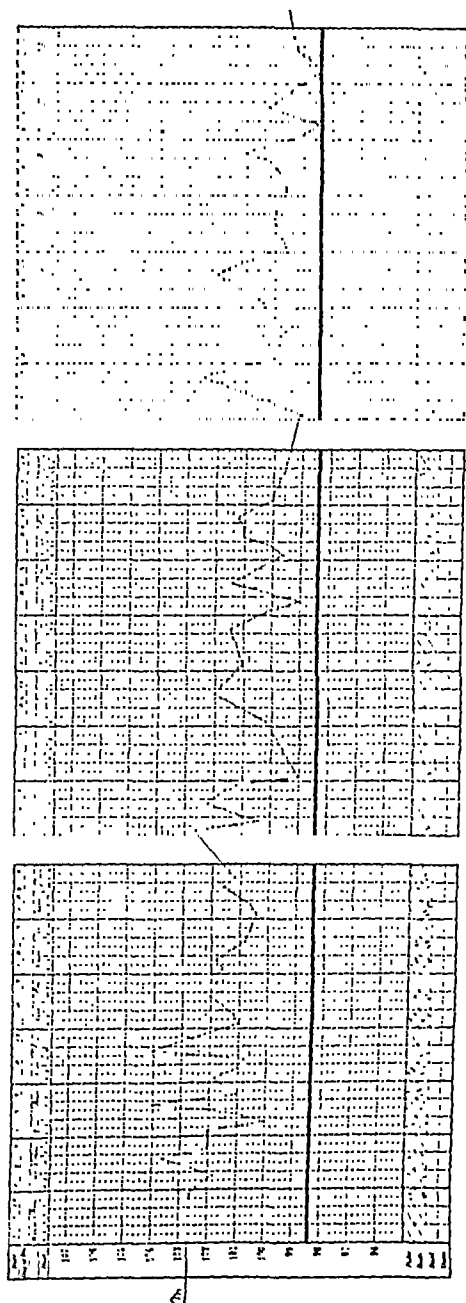
Fig. 41.—Case I.   Temperature curve.

On the eleventh day after her admission, practically the end of the third week of her illness, there were impaired resonance,

Fig. 42.—Radiograph of Case I. Right-sided posterior mediastinal pleurisy previous to puncture.

decreased fremitus, and tubular breath sounds over the right lower posterior. Aspiration was done, but with a dry tap. Fluoroscopic examination made at this time revealed no fluid

in pleura, but "findings suggest changes due to 'flu' congestion of right lung."

Fig. 43.—Same case as Fig. 42 after period of about ten weeks.

Clinical notes, made from time to time, show some improvement up to February 28, 1919, when she was transferred to the Medical Service, Ward 61, on the diagnosis of convalescent influenza and pleurisy.

Symptoms and physical findings on admission to Medical Service: Except for slight pain in right chest and occasional dry cough, there were no symptoms. Physical examination revealed dulness, decreased fremitus, and distant tubular breath sounds, without râles, over right lower posterior, close to the spine in the phrenovertebral angle.

Laboratory Findings.—*Roentgen Examination.*—"In the posterior mediastinum on the right side in its lower portion there is a large bulging shadow of lesser density than the heart, which has the appearance of an encapsulated mediastinal effusion. The balance of the pulmonary shadows on both sides show distinct increase of peribronchial tissue."

Sputum.—Unsatisfactory specimens were negative for tubercle bacilli.

Urine.—Trace of albumin, but no casts.

Blood.—Hemoglobin 60 per cent. Leukocytes 8800. An exploratory puncture was made in the right tenth intercostal space about $2\frac{1}{2}$ or 3 inches from the spinous processes, over the area of physical findings. About 10 c.c. of thick, creamy pus was aspirated, which on examination revealed many pus-cells, but no organisms. No attempt was made to remove more than the amount given, but two days later a therapeutic aspiration was attempted without success. No fluid could be obtained.

Subsequent Roentgen examination was made with similar findings, which gradually lessened, with the physical signs, until April 23, 1919, at which time she had practically recovered and was discharged.

CASE II

Female, age twenty, admitted August 25, 1919, and sent to Medical Service on the diagnosis of lobar pneumonia.

History on Admission.—Until August 21st was perfectly well, when she did not feel able to go to work and remained in bed, although she had no definite complaint. Had no chill, pain, or discomfort. Felt weak and tired. Occasionally had a slight, non-productive cough, which had been present for several winters.
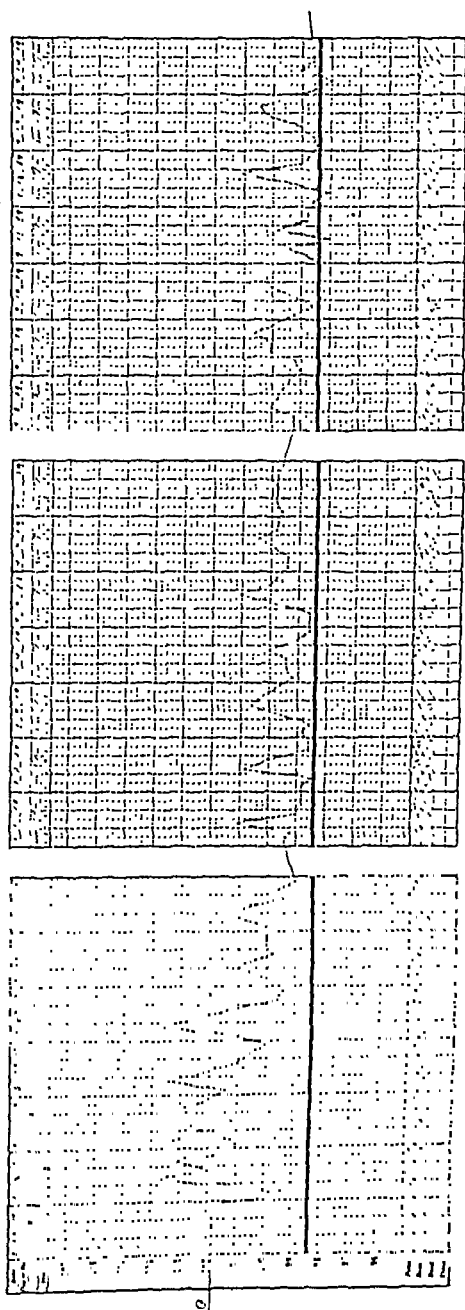
Fig. 44.—Case II. Temperature curve,

Family and Personal History.—Former negative, as also the latter, except for attack of flu November 18, 1918, which was moderately severe, with a protracted convalescence.

Fig. 45.—Radiograph of Case II. Pseudopericardial type of mediastinal pleurisy.

Physical Findings.—Examination was negative except for the chest. Posteriorly over the lower half of either lower lobe

along the spine there could be elicited a triangular area of dulness, the base downward, and about 2 inches in width. Over these areas the tactile fremitus was decreased, while on ausculta-

Fig. 46.—Same case as Fig. 45 during convalescence.

tion a marked bronchial breathing with a few crepitant râles on forced inspiration could be heard.

Laboratory Findings.—*Roentgen Examination.*—"A large

effusion is present in the lower posterior mediastinum." Three subsequent examinations confirmed above findings.

Sputum.—Eight recorded examinations were negative for tubercle bacilli. Pus-cells and mixed type of organisms were numerous.

Urine.—Repeated examinations were negative.

Blood.—August 26th: Hemoglobin, 95 per cent.; reds, 5,120,000; whites, 15,000. September 4th: Hemoglobin, 90 per cent.; reds, 5,440,000; whites, 16,000. September 10th: Whites, 14,500.

September 1st an exploratory puncture was made in the right ninth intercostal space posterior, over the area of maximum tubular breathing, and 2 or 3 drops of pus removed. Further efforts were unsuccessful. Microscopic examination revealed many pus-cells and cocci, single and in short chains.

Condition improved gradually, with decrease in physical signs and Roentgen findings, until September 23, 1919, when she had practically recovered and was discharged.

DISCUSSION

The first case was admitted as an influenza and developed symptoms and signs of a bronchopneumonia, from which she recovered, but subsequently presented physical findings which were interpreted as encysted empyema. After the Roentgen examination and report as given, it was a question if the effusion was in the anatomic posterior mediastinal space or was simply an encysted posterior mediastinal type of a pleurisy. Fluoroscopically it was easily demonstrated that the fluid was posterior to the heart shadow. Stereoplates confirmed this conclusion. Anatomically considered, an effusion into the posterior mediastinal space, while perhaps not impossible, is hardly likely. F. Barjou, in his "Radiodiagnosis of Pleuropulmonary Affections," translated by James Albert Honeij, gives a description of this condition, with some diagrammatic sketches, both of which will be given.

"Mediastinal pleurisy is rare. It may be purulent or serofibrinous. It may be dry without effusion. Of these different

forms, the first is much the most important and the most serious. Left to itself, it may result fatally. It necessitates, therefore, intervention, which may be successful, provided it is not resorted to too late. It is important to make the diagnosis as early as possible.

"The two other forms are less serious. They are ordinarily cured by medical means. In the dry form it is sufficient to apply a counterirritant over the inflamed zone, without any
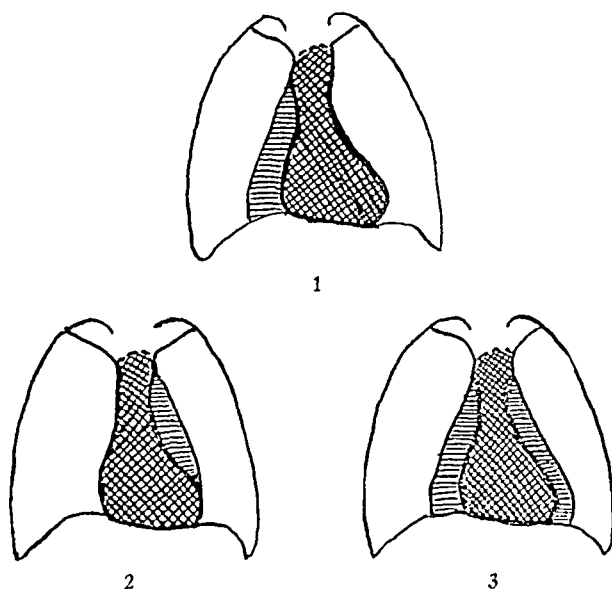
Fig. 47.—Mediastinal pleurisy: 1, Right mediastinal pleurisy. 2, Left mediastinal pleurisy. 3, Pseudopericardial form.

need of exactness. In the serofibrinous form puncture is rarely necessary; the cure is spontaneous.

"*Mediastinal Pleurisy With Effusion.*—The collection may be present in the anterior or in the posterior mediastinum; it may be unilateral or bilateral. It may be combined with diaphragmatic pleurisy or with pericardial effusion.

"What, in general, may be considered characteristic of radioscopic examination in mediastinal pleurisy is the fact that the

abnormal shadow which it reveals is always superadded to the median shadow. It deforms and increases this shadow at a point differing according to the location.

"In the posterior mediastinum it is seen as a dark band adherent to the vertebral shadow and occupying all the inferior part of the thorax from the hilus of the lung to the diaphragm. In the anterior mediastinum, on the right, it is seen as an obscure triangle, the apex of which corresponds to the hilus of the lung and the base to the diaphragm; on the left it is like an enlarged aortic shadow and surmounts that of the heart, as in aneurysm of the descending portion. Such, at least, is the theoretic description. In reality, things are not always as clear as that, and to detect mediastinal pleurisy it is necessary to compare the clinical symptoms with the radiologic indications."

The radiographs of the first case compare very well with the diagrammatic drawing of Barjou of a right posterior mediastinal pleurisy. Confirmatory proof was furnished by the puncture.

In the second case a diagnosis of posterior mediastinal pleurisy was made, on the physical findings, which 'was con-. firmed by Roentgen examination and puncture. The radiographs revealed a shadow similar to the pseudopericardial type as given by Barjou in his diagram.

CLINIC OF DRS. FREDERICK TICE AND VINCENT J. O'CONOR

WASHINGTON BOULEVARD HOSPITAL

RIEDEL'S LOBE OF THE LIVER SIMULATING RENAL TUMOR

THE identification of masses which are palpated on abdominal examination frequently necessitates very careful and detailed investigation before the true nature of the tumor is ascertained. In order that we may constantly bear in mind the possible sources of the more unusual tumors, that may be discovered on palpation of the abdomen, it is important that all conditions of this character be carefully recorded.

"Riedel's lobe," or linguiform lobulation, is a tongue-like elongation of the anterior margin of the right lobe of the liver. It is merely a prolongation of the right lobe itself, and is not a distinct lobe in the true sense of the word.

Linguiform lobulation has occasionally been found in young children of both sexes, but clinically and at necropsy it has been reported almost solely in women. Among those who have had the opportunity of studying this condition at autopsy there has been considerable discussion as to the etiologic factors concerned in its production.

A careful review of the development of the liver and its lobules in man reveals no morphologic reason for the occurrence of a linguiform lobulation.

Riedel, after whom the lobulation is named, originally described the condition in individuals with enlargement of the gall-bladder. He ascribed this prolongation to the gradual enlargement of the gall-bladder tending to cause downward traction on the anterior margin of the right lobe. Others have noted this elongation in association with gall-bladder disease.

A limited number of autopsy observations on the part of other investigators does not confirm this process of development of the lobulation. From the evidence at hand it would seem that it may occur as an anatomic abnormality, or that it may be acquired through conditions which tend to force the anterior margin of the right lobe downward.

The prolongation is always freely movable and the structure of the lobule is of normal hepatic tissue. The lobule proper is joined to the

Fig. 48.—Diagrammatic drawing showing anterior view of linguiform lobulation of the right lobe of the liver.

Fig. 49.—Posterior view showing linguiform lobulation of the right lobe of the liver anterior to normal kidney. The notch above the elongation being anterior and only slightly lower than the upper pole of the right kidney.

anterior surface of the liver by a narrow isthmus of hepatic tissue, the prolongation enlarging both anteroposteriorly and laterally just below this junction point. The mass is always felt in the

right side of the abdomen, with the upper border outlined below the margin of the liver at a variable distance. Owing to the posterior position and the narrowness of the isthmus, the anterior superior surface or "upper pole" of the mass is practically always considered the upper limitation of the mass, and its connection with the liver proper is not recognized.

Since the mass moves freely on respiration and the upper border is in the right lateral portion of the abdomen, with the mass extending below the iliac crest into the pelvis, the natural inference on physical examination is that the mass originates in the kidney substance. Two such cases were recently reported by one of us, and the statement was made that only by making a ureteropyelogram on the right could the true condition be ascertained.

Linguiform lobulation causes no subjective symptoms, and it is usually discovered during an examination brought about by consultation for some abdominal complaint.

The recognition and differentiation of this mass is the more difficult depending on the severity of the patient's symptoms. In the presence of an acute infection in the urinary tract, with a marked rise in temperature and pain in the flank, the occurrence of a linguiform lobulation makes a differentiation very difficult, and the apparent indications are for an immediate surgical procedure. In the presence of symptoms referable to the large intestine, a neoplasm of this structure is first brought to mind. At other times a retroperitoneal tumor other than kidney may suggest itself.

Fortunately, the true nature of the mass may be readily discovered by studying the filling outline of the intestinal tract, or the draining portions of the kidney when they have been distended with fluid which is opaque to the x-rays. Either or both of these procedures should be utilized in determining the nature of any obscure mass in the abdomen.

The following case is most interesting and instructive:

Annie V., a Jewish housewife, aged fifty-five years, presented herself complaining of slight discomfort in the lower abdomen and a persistant constipation of a mild degree.

Family and past histories were unimportant. The patient had had the usual diseases of childhood, but had never suffered with typhoid, pneumonia, phlebitis, or other cardiac or gastro-intestinal trouble. There had been no operations performed and she had never been in any accidents of serious note.

Her habits and general health were normal, and she had not been in the habit of wearing tight corsets except on very rare occasions.

The present trouble was of three to four years' duration, and was characterized by a dull aching pain across the mid-abdomen with a sense of soreness on pressure. These pains were never severe in character, never colicky in type, and did not radiate into thorax, groin, or flank. The patient felt that there was a definite association between the presence of the discomfort and her more or less persistent constipation. There was no pain in the epigastrium and she had never been jaundiced.

For two years the patient had had an occasional slight increase in frequency of urination to four to six times a day, and once or twice at night. No dysuria, pyuria, or hematuria had ever been noted.

For the past eighteen months the patient had frequently resorted to cathartics of various sorts to assist in emptying the bowel.

Physical examination showed a fairly well-nourished woman without serious complaint.

The skin, head, eyes, ears, nose, throat, and glandular systems were normal.

The lungs were clear and resonant throughout, with normal expansion. No râles or other abnormal signs were present.

The heart was normal in size, with normal sounds, no murmurs or thrills.

Blood-pressure was 118 systolic and 80 diastolic. The spine and osseous system was normal. The abdomen was slightly tender over the entire lower portion, but there was no rigidity, spasm, or asymmetry.

The spleen and left kidney were not palpable. The liver appeared to be normal in size, and a smooth border could be felt just below the costal margin on the right side.

In the right abdomen two masses could be felt.   The upper mass was just below the edge of the liver in the right upper quadrant midway between the midaxillary line and the mid-abdominal line.   This mass could be felt more easily by bimanual palpation through the flank.   It was the size of a small orange, with a smooth rounded outline, and moved upward and away from the palpating fingers on respiration.

Just below this mass and not in any way connected either with it or with the liver was a tumor mass filling the entire right abdomen and extending below the iliac crest into the pelvis.   It was ovoid in general outline, the upper pole could be grasped by the flexed fingers and held in position during respiration.   The mass appeared to fill the entire flank and was not tender to bimanual palpation.   It was firm, there were no nodulations, and the consistency was everywhere the same.

The reflexes were normal.   The extremities and genitalia were normal.

The examination of the blood showed 90 per cent. hemoglobin, normal erythrocyte and leukocyte counts.

The urine was normal in every respect.

x-Ray pictures showed the outline of the two masses almost exactly as defined by palpation, with the lower mass extending well below the iliac crest on the right side.

The possibilities for the identification of this mass which then suggested themselves were as follows: The upper, smaller mass might be a distended gall-bladder, the lower pole of a normal kidney, or a retroperitoneal tumor.   The larger mass appeared to be an enlarged low kidney both from its position and shape.   The question of this mass possibly being a linguiform lobulation of the liver was discussed and cystoscopy advised.

Cystoscopy.—The urethra was normal.   There was very slight heaping up of the bladder mucosa over the lateral walls at the neck of the bladder, but no polypi or erosions.

The bladder capacity was normal and the entire bladder appeared to be normal in every respect.   The ureteric orifices

were normally placed and functioned normally with a clear efflux.

The right ureter was catheterized for a distance of 30 cm. without difficulty. The urine flowed in normal peristaltic

Fig. 50.—Plain roentgenogram showing normal kidney outline above and large linguiform lobulation of the right lobe of the liver extending below the iliac crest.

waves and proved normal on chemical, microscopic, and cultural examinations.

Ureteropyelogram was made on the right by injecting 9 c.c. of 15 per cent. sodium bromid solution.

The pyelogram showed the kidney to be in normal position,

with normal gross outline, and with a normal pelvis and calyx arrangement. This identifies absolutely the mass which was felt above as being a normal kidney.

Fig. 51.—Right ureteropyelogram showing definitely a normal right kidney above and distinctly separate from the abnormal lobulation of the liver.

The lower mass, which casts the elongated shadow projecting below the crest of the ilium, is obviously a linguiform lobulation of the liver.

CLINIC OF DR. CHARLES A. ELLIOTT

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THE MANAGEMENT OF LEUKEMIA

"RADIUM Treatment of Leukemia; One Case with Sple-
nectomy" was the title of a clinic held by the author and pub-
lished in the Medical Clinics of North America in March, 1918.
At that time several cases of leukemia were reported, special
emphasis having been placed on the treatment with radium.
Since that time the patients who were presented have succumbed.
I wish to take this opportunity of giving a further and com-
plete report of the clinical course of one of the patients who
was observed constantly for three and a half years until the
time of her death; and to discuss the relative values of Roentgen
rays, radium, and splenectomy in the management of myelog-
enous leukemia.

Summary of Clinical Course.—Mrs. E. H. O., age thirty-
five years, was first admitted to Wesley Memorial Hospital
June 17, 1917. She received further hospital treatment on
numerous occasions and was constantly under observation until
her death on November 6, 1920.

Symptoms on admission were characteristic of myelogenous
leukemia, namely, general weakness, dyspnea, loss of weight,
attacks of jaundice, nausea and vomiting, nocturia, and a mas-
sive splenic tumor, with considerable pain in this region. The
mass in the abdomen was first noticed in June, 1916. At the
same time she noticed that her color was icteric, ashen. The
tumor mass grew rapidly until September, 1916, at which time
the lower pole rested on the pelvis to the right of the median

line above Poupart's ligament. For a number of months numerous tumors had been observed occurring in crops, widely distributed in subcutaneous tissues, and disappearing spontaneously after several weeks' duration. Headaches were severe; she had never had them before. Menses had been irregular for a year.

In 1914 she had had a single acute attack diagnosed as renal colic. In September, 1916 she became jaundiced and had severe vomiting. The family history is unimportant.

Physical examination on admission revealed a well-nourished woman of thirty-five years; numerous subcutaneous infiltrated masses 3 to 6 cm. in diameter, surrounded by hemorrhagic pigmented skin, were scattered over the body. She was subicteric, anemic; clusters of glands were just palpable in cervical and axillary regions and groins; no tenderness over the long bones. Lungs were negative. A systolic blow was heard over the apex of the heart; pulmonic second sound accentuated; otherwise normal cardiac conditions. A prominent mass, evidently a splenic enlargement of maximum grade, filled a large part of the left abdomen; the surface was irregular; three distinct notches present; no friction-rub or bruit. The liver was large, extending to the level of the navel; the edge was sharp, tender; one area, the size of a dollar, projected above the general level on the anterior surface. Reflexes normal. Pelvic examination showed normal conditions; the spleen was not palpable vaginally.

Blood examination was typical of myelogenous leukemia. Red blood-cells, 3,648,000; white blood-cells, 311,700; hemoglobin, 75 per cent.; polynuclears, 40 per cent.; lymphocytes, 8 per cent.; large mononuclears, 10 per cent.; basophils, 6 per cent.; eosinophils, 4 per cent.; neutrophilic myelocytes, 22 per cent.; basophilic myelocytes, 10 per cent.

Radium treatment was instituted; 2700 milligram hours of radium were applied over the spleen from June 19 to June 21, 1917. On July 13th the patient stated that she felt considerably relieved of the abdominal distention and pain; the spleen was definitely smaller; the white blood count was 176,000.

July 13 to 15, 1917 the second treatment of radium was applied over the spleen; 3800 milligram hours were given.

On July 21st the patient suffered an attack of acute epidemic parotitis; temperature was 103° F.; she was very ill for a few days, but recovered rapidly. On August 4th Mrs. O. reported feeling quite well; the white blood count had dropped to 15,950 and the spleen was greatly reduced in size; the lower pole was readily palpable, extending 4 cm. below the rib edge.

About three weeks following each of the above radium treatments superficial radium burns made their appearance at the sites of application. These healed slowly and were accompanied by much skin itching.

On December 1, 1917 the patient's general health appeared excellent; she had gained 34 pounds in weight; the spleen was greatly reduced in size, the lower pole being just palpable below the rib edge. White blood count 11,300. Menstruation had become normal. From December 1st to 3d a fourth radium treatment over the spleen was given.

Fearing a return of the splenic tumor, with its accompanying pain and distress, the patient consented to have a splenectomy performed. This was done by Dr. H. M. Richter on December 29, 1917, without incident.

During the following year (1918) no extended record of her case was made; in general, she experienced little discomfort. Blood-smears indicated the presence of leukemia, but the white count remained relatively low and the patient felt well. On February 15, 1918 the white count was 25,100. By June 11, 1919 the count had increased to 128,000, with 48 per cent. myelocytes. Radium was again used. During the remainder of 1919 radium was applied frequently in large amounts over the body—the long bones, shoulders, and ribs. The total amounts were not always accurately noted; 17,400, 10,500, 16,124 milligram hours were applied at various times. Following these periods of radiation a definite drop in the white count to as low as 75,000, 45,000, and 56,000 was noted. After each treatment the patient experienced considerable reaction, much

depression, headache, and nausea. It was evident that the same reaction occurred when the radium was applied anywhere over the body as had formerly occurred when the radium was applied only over the spleen.

With the progress of the disease it became evident that radium would have to be applied frequently in order to keep the white count within reasonable limits. During all of this time the patient felt and looked well; she had gained strength; she menstruated normally.

From March, 1919 to March, 1920 frequent attacks of severe headaches associated with nausea and vomiting were experienced; these attacks were periodic and not relieved by doses of morphin as large as $\frac{1}{2}$ grain hypodermically; they were interpreted as due to leukemic infiltration in the brain or, perhaps, due to intracranial leukemic tumors. March 13, 1920, after having had unusually severe headaches, it was decided to change from radium to x-ray therapy.

A series of x-ray treatments were then given until October 30, 1920 by Dr. Hollis E. Potter. At the beginning of this series the white count was 474,000; by April 22d it was 55,200. In June the count had increased to 203,000. After that it was never completely controlled.

On November 5, 1920 the patient was taken acutely ill, with delirium, temperature 105° F., retinal hemorrhages, cyanosis, tachycardia, incontinence. Bronchopneumonia developed. White blood count 448,000. She died after several days with manifestations of acute leukemia associated with an acute terminal infection.

A necropsy was performed by Dr. J. P. Simonds on November 6, 1920. A typical picture of myelogenous leukemia with terminal bronchopneumonia was found. There was red marrow in the shafts of the femur and the sternum; general lymph-node enlargement in the mesentery, along the aorta, and at the hilum of the liver; old healed tubercles in the pleura of the left lung with chronic adhesive pleuritis of the left pleura; fatty degeneration of the heart muscle with subpericardial hemorrhages; marked distention of the right auricle; goose-fat clots in heart.

The liver was enlarged, leukemic infiltration throughout; chronic cholecystitis and cholelithiasis present; chronic adhesive peritonitis; kidney showed multiple arteriosclerotic scars; stomach showed punctate hemorrhages in the submucosa. Pelvis was normal. The brain was not examined.

The blood-vessels in sections from all organs contained great numbers of leukocytes, most of which were myelocytes. Sections of blood-clots from the heart were composed chiefly of leukocytes. Lungs and liver showed very marked myelogenous infiltration. The masses found along the aorta and at the hilum of the liver were lymph-nodes, the lymphadenoid tissue of which had been more or less completely replaced by tissue resembling that in the marrow of the femur, which was richly cellular and contained many nucleated red cells and myelocytes. The same was true to a less extent of the lymph-glands from the mesentery. The lungs showed a hemorrhagic bronchopneumonia.

Outline of Treatment.—July 24, 1916, almost a year before the patient came under our observation, the spleen was of maximum size; weight 100¾ pounds; red blood cells, 3,900,000; white blood cells, 480,000; hemoglobin, 50 per cent.

June 18, 1917 there was a white count of 311,200. June 19th, 20th, and 21st the *first applications of radium* were made over the spleen, a total of 2900 mg. hours; one week later the white count was 134,000; the spleen had decreased in size. The *second applications of radium*, totaling 3800 mg. hours, on July 13th, 14th, and 15th reduced the white count to 19,000 after six days; the spleen was much decreased in size; the patient felt well and had gained 9 pounds in weight. During the remainder of July and in August the count was under 20,000; September 5th it was 30,200. On September 5th and 6th a total of 1800 mg. hours of *radium* was applied over the spleen; one week later the count was 16,300; the patient had gained 20 pounds in weight. Just prior to the *fourth application of radium*, a total of 2300 mg. hours on November 20th, 21st, and 22d, the white count was 26,800; three weeks later it was 10,000. *Splenectomy* was performed on December 29, 1917;

sixteen days later the patient was discharged from the hospital in good physical condition; white count 12,100.

During 1918 the white count was controlled and the patient kept apparently in good health by *five series of radium applications* on February 18th, 600 mg. hours; July 8th, 960 mg. hours; July 13th, 960 mg. hours; August 24th, 700 mg. hours; and October 27th, 1600 mg. hours. The radium was applied over the long bones, sternum, and ribs.

January 11, 1919 the white count was 128,000. *Radium* applied on three successive days produced drowsiness, fever, exhaustion, diarrhea, and nausea. White blood cells 147,000 on January 25th; 171,000 on February 15th; 152,000 on March 1st; 180,000 on March 19th. A total of 17,400 mg. hours of *radium* was used on March 24th, 25th, and 26th. On April 12th the count was 142,200; on May 2d, 75,000. By July 3d the number of white cells had increased to 240,000. July 3d to 7th *radium* (10,500 mg. hours) was employed; a severe reaction followed; eleven days later the white count was reduced to 45,000. October 11th there were 252,000 white cells; December 5th there were 324,000. *Radium* (16,124 mg. hours) used on December 5th, 6th, and 7th on various parts of the body, without regard to particular application over bones, produced a marked reaction—headache, drowsiness, nausea, and edema. December 13th the count was 82,500; December 27th, 56,000. The patient felt well.

By February 21, 1920 the white cells had increased to 324,000; by March 13th, to 474,000. Because radium in the last applications had produced severe although temporary reactions as well as slight burns without controlling the blood count, as it had done at the outset, Roentgen-ray therapy was recommended. Thirteen series of radium applications had been employed. The patient was referred to Dr. Hollis E. Potter, who administered, in all, *nineteen treatments with Roentgen rays* over long bones of extremities, shoulders, hips, and thorax. On April 2d, a week after the third treatment, the white count was 119,200; on April 22d, five days after the sixth treatment, it was 55,200. May 22d, two weeks after the eighth treat-

ment, the count was 107,400; June 29th, after the twelfth treatment, it was 166,200; July 28th, a week following the fourteenth treatment, it was 195,000. In September the patient developed severe attacks of neuritis in the right arm, shoulder, and back. September 25th, three weeks after the sixteenth Roentgen-ray exposure, the count was 90,800. On November 5th she was admitted to the hospital, delirious. febrile, with signs of bronchopneumonia; white blood-cells 422,000. *Radium* was applied at various sites without effect. The patient died November 6th.

Conclusions.—The case presented is one of chronic myelogenous leukemia of about four and a half years' duration. Under the management described the patient, first seen about one year after the onset of the disease, was relieved of symptoms for more than three years. In view of her grave condition at the time of our first examination it would seem fair to assume that a prolongation of her life for three and a half years was effected.

Radium gave prompt, unmistakable, and marked relief, as indicated by decrease in white blood count and in size of spleen, by subjective relief from symptoms and by increase in strength and weight. The patient was able to take up her usual duties apparently in good health.

Splenectomy afforded relief from a previously uncomfortable tumor mass. It had no other evident effect on the course of the disease. Following the operation the application of radium elsewhere over the body surface produced the same effect as had been produced by radium applied over the spleen.

Radium, used at intervals determined by the white blood count, seemed to forestall the disease in so far as clinical manifestations were concerned for about three years, when severe headaches. ascribed either to leukemic infiltration or to actual leukemic tumors in the brain, appeared. The early treatments with radium (for about two years) were followed by definite improvement in general health and by marked reduction in the white count; later applications were less effective and were followed by depression and persistent headaches.

Roentgen-ray treatment, instituted because of the persistence

of headaches in spite of radium applications and because of periods of depression following radium therapy, produced the same effect on the blood-picture. No marked quantitative or qualitative difference in the response to these agents was evident.

The terminal event—acute leukemic manifestations with pneumonic infection—was that frequently observed in leukemia regardless of the method of treatment.

CLINIC OF DR. CHARLES LOUIS MIX

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MEDIASTINAL SARCOMA AND METASTASIS IN THE LEFT LUNG

THE patient is sixty years of age, by occupation a policeman. He entered the hospital March 30, 1921 with the following chief complaints: First, sores in the skin of the left armpit and of the left side of the chest wall; second, loss of weight; third, loss of appetite; fourth, weakness.

He noticed that eight months ago there appeared in his left armpit two or three inflammatory nodules, seemingly to contain pus and having a bluish color. These nodules were very much inflamed, but were not so sore as boils ought to be. These inflammatory nodules which appeared in August, 1920 disturbed him to such an extent that in the end of December, 1920 he went to a neighborhood physician, who opened the nodules under gas. After they had been opened they did not heal, but have been discharging pus and serum ever since.

At the time of the appearance of the sores in the left armpit the patient began to notice that he was losing in weight. Previous to this he had always been healthy. He had been a police- man for thirty-five years and had never missed a day's work. He is 6 feet, 2 inches tall and weighed 285 pounds. Between last August and the present time, a period of about eight months, his weight has fallen about 110 pounds, so that now he weighs 175 pounds. Two months ago he began to lose his appetite, and on that account went to Hot Springs, Arkansas, hoping he would be benefited by the treatment there. He remained at the springs a month, gradually growing weaker and con- tinually losing flesh.

His only complaint aside from his loss of weight and loss of appetite is his weakness. He does, however, admit that a month ago (February) nocturia appeared. He says it was necessary for him to rise several times during the night, and he passed a good deal of water. Of late he gets up because he thinks he ought to, not because of any desire. There is no burning on urination or any incontinence. Of late he has noticed that the urine has been of high color.

His family history is negative. His mother is still living; his father died of accidental death. He has two brothers living and well; he has no sisters. No tuberculosis or carcinoma occurs in his family history. There is no history of gonorrhea or syphilis. He is the father of 3 children now living and well, and of one girl who died when she was a child. His habits have always been regular, he has never been a drinking man, and there is nothing about him to indicate that he is not telling the truth.

On examining the patient physically the most notable thing about him is his intense asthenia and the external appearance of weakness. Although he walked into the hospital, he seemed scarcely able to do so, and sank into a chair at the first opportunity. Looking at him one also was immediately struck by the evidently great emaciation which he had undergone. One felt instinctively that he must be suffering from some malignant disease and one would confidently expect to find the focus very easily.

Very noticeable also is the appearance of his skin. It is exceedingly dry and itchy and as scaly as the skin of a fish. These scales are particularly marked all over the abdomen and on the back. He says his skin was never in this condition until the trouble which came upon him brought it about. He also is somewhat dyspneic. On entrance his pulse was only 68, but that was because it was not accurately recorded by the nurse. The next time it was taken it had risen to 104, and the following day varied from 90 to 100. This dyspnea was evidently due to myocardial weakness because it was associated with a slight degree of swelling in both of his lower extremities.

As time went on during his stay in the hospital the swelling became greater, and subsequently he developed a mild ascites.

This accumulation of fluid was evidently due to cardiac insufficiency rather than renal. For example, urinalysis of a single specimen made the day after his entry showed a specific gravity of 1034, alkaline reaction, and the absence of albumin, sugar, red blood-cells, and casts. The twenty-four-hour specimen collected during the next twenty-four-hour period amounted to nearly 500 c.c., the specific gravity 1026, the reaction alkaline, and it also was free from sugar, albumin, casts, or red blood-cells. Four more urinalyses were made on April 11th, 13th, 15th, and 18th. These were all normal. Moreover, his blood-pressure was only 120 systolic and 80 diastolic.

Examination of the lungs was not entirely negative. He had a cough and spat up sputum which contained pus-cells. Four slides were examined on four successive days for possible evidence as to the cause of the pulmonic disturbance. No tubercle bacilli were found, but the pneumococcus, staphylococcus, and streptococcus were present. One slide in particular showed a large number of polymorphonuclear cells with only a few epithelial cells, thus indicating an infectious process going on somewhere within the lung. From the external examination, however, nothing could be found on percussion or auscultation outside of bronchial râles in the bases of both lungs.

Examination of the abdomen revealed nothing but a small amount of ascitic fluid, evidently due to a weakened heart. The liver was not enlarged nor was it atrophic. The spleen could not be felt and there was no evidence to lead one to the conclusion that it was in any way implicated. His bowels move regularly without a laxative. The stools are never black, clay-colored, or abundant (hemorrhage, biliary tract blockade, pancreatic disease). No tumor mass could be felt in the pancreas. There was no fat in the stools and no sugar on urinalysis. The gall-bladder was never sore when palpated and there was no jaundice or history of jaundice. There were no enlarged glands in the neck or either armpit or in the groin. There were no enlarged nodules on the surface of the chest, such as occa-

sionally show in carcinomas. The rectal examination proved quite negative.

Especial attention was paid also to the examination of the stomach. Nothing was found abnormal in the usual tests, both laboratory and *x*-ray. Nevertheless, it was felt that there must be some malignant tumor about him. Having exhausted all the ordinary tests in the search for carcinoma—namely, of fauces, throat, esophagus, lungs, stomach, both pyloric and cardiac ends, pancreas, cecum, sigmoid, rectum, prostate—it was felt that there still must be some focus somewhere in the body.

The sores in the skin of the left armpit and on the side of the trunk were extremely peculiar. A very little thin pus exuded from them. This pus was sent to the laboratory and examined for any possible parasites. No blastomycosis, sporotrichosis, or other forms of parasitic fungi could be found. The patient constantly ran a temperature which varied from normal to 100.6° F., and on some days the temperature would not go above 99.2° F. On other days it would rise to about 100° F.

Blood-cultures were frequently made and also proved negative. The blood examination showed no characteristic changes. The hemoglobin on entrance was 62 per cent., red cells 3,345,000, but a differential count of 10,600 white cells showed a polymorphonucleosis of 92 per cent., with only 6 per cent. of small lymphocytes and 2 per cent. of large mononuclears. This count was repeated in order to make certain, and practically the same results were obtained, namely, 93 per cent. polymorphonuclears, 6 per cent. small lymphocytes, 1 per cent. large mononuclears. These counts were made by two different individuals, so we have reason to think they are quite correct. The blood Wassermann test was negative, so that the ulcerations beneath the left arm could hardly be regarded as due to any specific infection, which he furthermore denied. The polymorphonucleosis was rather puzzling and suggested the possibility of sarcoma somewhere within the body, yet no sarcoma could be found.

During April the left hand, then the wrist, forearm, and

arm became edematous. This edema suggested the possibility of some interference with the return flow of venous blood from the arm to the body. There did not seem to be a lymphatic blockade because the edema was entirely confined at first to the hand and wrist. Gradually the edema crept up the arm, and it was this edema which determined the making of a skiagram of the chest. It was felt that the interference with the return flow of venous blood must be consequent upon some disturbance in the mediastinum. The swelling of the upper extremity when not due to lymphatic blockade, as in carcinoma of the breast, or thrombophlebitis, as in infection, is very apt to be due to a mediastinal tumor with pressure upon the subclavian or innominate vein, or both.

Notwithstanding the negative physical examination of the chest the skiagram was ordered, largely because of the polymorphonucleosis, pus in the sputum without tubercle bacilli or signs of abscess, and the presence of the swelling in the left arm. The plate of the skiagram shows rather vaguely a widening of the upper portion of the mediastinum and a well-defined tumor in the left lung. This tumor is evidently metastatic from the mediastinum, since it is separated from this by an interval of clear lung. If the mediastinal tumor is, as most mediastinal tumors are, a sarcoma, and it probably is because of the differential white count, already the cells have broken into the venous circulation and have reached the right side of the heart, and from there have been carried into the lung itself, where they have started the growth (Fig. 52).

The origin of this mediastinal growth, which the x-ray alone discloses, is not apparent. It seems to be a primary mediastinal sarcoma with secondary sarcoma in the lung. We cannot make out a relationship between the sores in the armpit and the present mediastinal growth, yet there may be a relationship. There is one sure clue which the patient has given to us. He says that the nodules which were inflamed and which seem to contain pus had a bluish color associated with the red of an inflammation. This makes one wonder whether these nodules which were removed were primary cuta-

neous sarcomatous masses. We cannot at present detect from superficial examination of these sore spots any pigmentation; and I have asked Dr. E. Wyllys Andrews to look at the patient with a view to determining whether the suppurative areas have anything to do with the mediastinal growth. Unfortunately, neither he nor I are at the present time able to demonstrate any such relationship. We have, therefore, to demonstrate
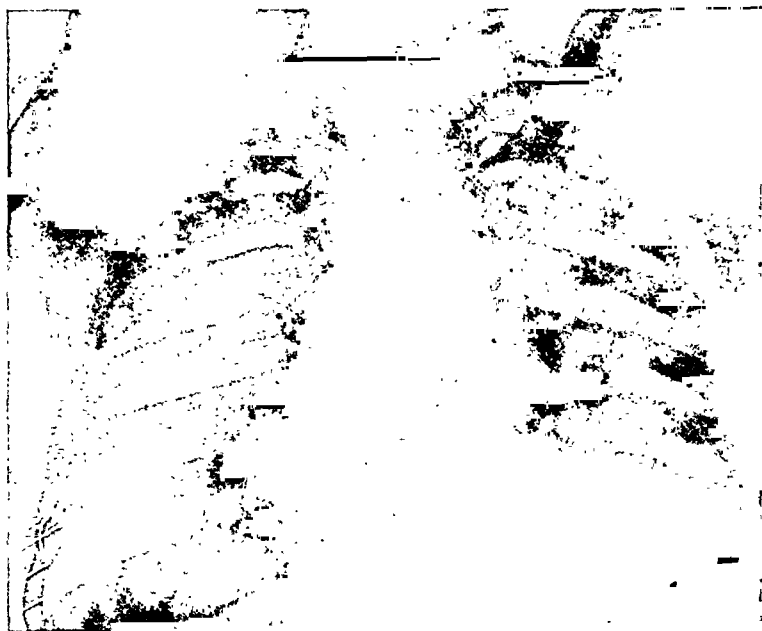
Fig. 52.—Mediastinal tumor (melanosarcoma) with metastasis in the left lung opposite the fourth, fifth, and sixth dorsal vertebræ.

the case as one of probable mediastinal sarcoma with secondary metastasis in the right lung, this metastasis giving no physical signs whatever, but being found only by x-ray examination.

The report by the radiographer shows an increase in the density of the hilus of the left lung. This area is surrounded by an area of deepened shadow, and has some of the characteristics either of a new growth or of a chronic inflammatory infiltration. The tumor mass in the lung is perfectly apparent.

wo other x-ray plates from a similar case of mediastinal
r with secondary tumor in the lung appear in the shadow-
The tumor seen in Fig. 53 shows a rounded black shadow
een the fifth and sixth ribs in the right side, apparently
ected with the mediastinum.   In order to demonstrate

Fig. 53.—Dr. Sloan's case, seen with him recently at the Illinois Central
ital. The tumor shows as a rounded black shadow between the right
and sixth ribs, apparently connected with the mediastinum.

connection an artificial pneumothorax was made by Dr.
n, and the tumor could be seen collapsed against the medi-
ıum. This patient is still living (Fig. 54).
igure 55 shows a tumor in the right lung, also connected
the right mediastinum, and a right-sided mediastinal

tumor. We watched this tumor grow in the plates till it led to the death of this patient in March of this year.

Returning now to the case being reported, there remains to be added merely the fact that the patient suffered a great deal from his feelings of weakness, the swelling and stiffness

Fig. 54.—Same case with artificial pneumothorax to demonstrate the relationship suggested in Fig. 53. Note the very well-defined tumor collapsed against the right mediastinum.

in the left arm, and the coughing, until finally he died on April 19th at 9 A. M. The previous day he had been very weak and drowsy without definite pain, but with involuntary movements of the bowel for the first time during his stay in the hospital. His pulse at 9 P. M. the night before his death was 104 and his

temperature 98.6° F.   At 4.30 A. M. his respirations began to be quite labored.   He was very restless and irrational and his pulse was 160.   At 6 o'clock his respiration rate was 30 and his pulse 120.   At 9 A. M. respirations gradually ceased.

Postmortem examination was made.   The heart was negative except for more than the normal amount of pericardial fluid.   The abdominal cavity showed a moderate ascites of

Fig. 55.—Tumor of the chest, connected with a mediastinal tumor.   This tumor has already proved fatal.

cardiac and not of portal origin.   The intestines, both the large and small bowel, were in every way negative.   The stomach was somewhat dilated, but the pylorus and the cardia showed no signs of carcinoma.   The liver was normal.   The gall-bladder was distended and incompressible, but there were no stones in it, and there was no demonstrable pathology of its walls.   There was no apparent blockade of the cystic duct notwithstanding the gall-bladder distention.   The pancreas

was normal. There were no nodules to be found either in the head, body, or tail. Both kidneys were normal and the spleen was not enlarged. No tubercles could be found on the peritoneum and no metastatic nodules of any sort were found within the abdominal cavity. The rectum, cecum, and sigmoid were normal. There was no abnormality of the bladder or of the prostate.

The lungs were then removed from the thoracic cavity. There were no adhesions of the pleura on either side. There was no great increase of fluid in the pleural cavities, though perhaps there was some slight increase. On removing the whole of the lungs and mediastinum *en masse* it was found that the mediastinum was larger than it should be. Sectioning the mediastinum, three or four large melanosarcomatous masses were found in it, and the area of the right lung which showed in the skiagram was also the site of a partially broken-down and suppurating melanosarcoma, from which the pus-cells found in the sputum were derived. There was nothing upon the patient's body to indicate the origin of such a tumor until the axillary tissue was removed from the left armpit. Then there was found beneath the ulcerated areas a diffuse melano-sarcomatosis through the axillary fold. Curiously enough, it had not produced any hard masses, so that to the feel no distinct tumor could be made out either by Dr. E. Wyllys Andrews or myself, and yet this diffuse sarcomatous mass entirely permeated what remained of the axillary cavity; and islands of similar tissue were found along the inner side of the left arm beneath the suppurating spots.

This case is merely another of the very large number in which a fatal termination has been quickly brought about by interfering with a melanotic tumor. Whether the melanotic patches in his left armpit which the patient called suppurating nodules had already broken into his general circulation when he was operated upon in December is difficult to tell. At any rate, it is a fact that the operation undoubtedly hastened his death by leading to a dissemination of the disease. Leave pigmented moles alone!

THROMBOSIS OF THE BASILARY ARTERY, WITH A CLINICAL PICTURE SUGGESTING ENCEPHALITIS LETHARGICA

THE patient was a young woman twenty-nine years of age. She entered the hospital March 27th with an axillary temperature of 100.2° F. and a pulse of 100. She was moaning continuously and unable to give any account of herself both by reason of her irrational state and her inability to articulate. Her husband stated that the trouble began two weeks before with severe headache. She had been accustomed at times to headache, but never in her life had she had anything so severe. The headache was neither nocturnal nor vesperal, but occurred all day long and all night long. After persisting with considerable intensity for five days the headache subsided in part until two or three days before admission, when it began again with great violence. The patient then for the first time began to show some deviation of the eyeballs. She next became delirious, then incoherent, and finally unable to articulate. All of this came on within a few hours' time, so that she was brought into the hospital in great haste, with the probable diagnosis of encephalitis lethargica.

She was examined rather carefully the morning of March 28th, and at that time there was pupillary inequality, the right pupil being larger than the left. There was also internal strabismus, both eyes turning slightly inward, due to a paresis of both external recti. There was also a slight paresis of the left side of the face. Observe that these cranial nerve involvements suggest medulla and pons. It was impossible for us to get the patient to protrude her tongue, so it is impossible to say whether there was a paresis of one or the other side. We cannot then say definitely as to whether one or other of the hypoglossal nerves are involved. The patient was semiconscious,

could be roused, but was unable to utter any words. This dysarthria suggested a pontine lesion. Both knee-jerks were exceedingly exaggerated. The same was true of the Achilles' jerks. There was both bilateral ankle-clonus and patellar clonus. Moreover, there was a good deal of tremor all over the body. The Babinski reflex was present in both great toes. There was rigidity of both of her legs and of both arms. The superficial reflexes, the epigastric and hypogastric, were wanting. The sensory disturbances could not be made out because of the patient's condition.

In the presence of such findings and with such a history a diagnosis of encephalitis lethargica could well be made. The disease began with an extremely severe headache and progressed rapidly with signs of basal disturbance, as indicated by the behavior of the pupils, which were inactive to light, and of the eyeballs and the face. The· headache alone was indicative of a meningeal disturbance. The paralysis of the extrinsic muscles of the eye and of the face were evidence of disturbance at the base of the brain. They might be looked upon as basilary, meningitic, or as due to a disturbance of the gray matter possibly in the floor of the aqueduct of Sylvius, though the third and fourth nerves seemed intact, and extending back to the nuclei of the sixth and seventh nerves, as in ordinary encephalitis lethargica. The behavior of the knee-jerks indicated an irritation of both pyramidal tracts, and seemed, if anything, to be too pronounced for an ordinary case of lethargic encephalitis. Due to the patient's semicoma and dysarthria no information could be obtained as to sensory disturbances or the condition of the fibers of the fillet.

During March 28th her temperature varied between 99.6° and 100.2° F. in the axilla, and respirations varied from 36 to 50, and pulse from 96 to 122. Though she voided her urine spontaneously during the first twelve hours, she quickly lost control of the sphincter and had to be catheterized at 4.30 P. M.. and from that time on at times.

Further evidence of pontine trouble developed. For example. about noon of March 28th she developed a good deal of difficulty

in swallowing, and later in the evening the respirations became labored at intervals, with intervals of comparatively quiet respirations intervening. It seemed as though there was some influence of varying degrees of intensity being exerted upon the respiratory center by reason of some inflammatory process going on in the medulla. The following day, March 29th, her temperature varied between 100.2° F. at 9 A. M. and 104° F. All temperatures taken on this day were either axillary or rectal. The respirations did not vary further from 36 to 40 until 7 P. M., at which time the respiratory rate suddenly increased to 52 at the same time that the axillary temperature was 104.° F.

Very peculiar was the behavior of the pulse during the 29th. It showed a marked degree of pneumogastric inhibition, varying from 72 to 92; at one time when the temperature was 103° F. the pulse was 80; at another time with a temperature of 102.8° F. the pulse was 72, and at 10 P. M., when the temperature was 104° F. in the axilla, the radial pulse was only 84. Such a pulse variability, which occurs in meningitis, must be looked upon as due to intense stimulation of the pneumogastric inhibitory apparatus. It usually means an irritating process affecting the nucleus ambiguus, and was so interpreted by us in this case. On March 30th the same discrepancy between the temperature and pulse was maintained; for example, at 7.30 A. M. the respirations were 42, the rectal temperature was 104° F., and the pulse was 76. During the 30th her temperature remained high, ranging from 102.2° to 104° F., the respirations varied from 32 to 48, and pulse from 76 to 104.

On the 30th there was a marked change in the reflexes. Whereas before there had been extremely lively knee- and ankle-jerks, with ankle- and patellar clonus, on the 30th the patellar and ankle-clonus disappeared, leaving only an increased knee-jerk with a double Babinski toe-phenomenon. Such a reduction in patellar and ankle-clonus argued either for improvement or for a paralytic succeeding an irritative stage.

During the 30th her respirations varied from 32 to 52 and at times were very labored. On March 31st the patient grew rapidly worse. At 1 A. M. her temperature was 104.8° F.

axillary, the pulse was 94, and the respirations 50. She was sweating profusely. She seemed comfortable, but was so stuporous that it was impossible to say very much about her. At 5 A. M. her rectal temperature had risen to 105° F., her respirations to 56, and the pulse remained at 96. At 6.30 her pulse had risen to 104, though the temperature had dropped to 103° F., the respirations remaining still at 56.

This increase in pulse-rate is ominous. It is such an increase as occurs in fatal cases of meningitis and marks the transition from irritation of the pneumogastric inhibitory apparatus to paralysis. In meningitis whenever the pulse begins to rise one can tell that the end is near, and ordinarily death takes place when the pneumogastric inhibitory paralysis supervenes, which is usually when the pulse reaches 160. Such proved to be the case with this patient. At 9 o'clock the pulse was 124, temperature 104° F., and respirations 64. At 11 o'clock her pulse was 146 and growing weaker; at 12 o'clock the axillary temperature was 105° F., the pulse 130, the respirations 64. From now on the pulse became much weaker and much more unequal and arhythmic. At 1 P. M. the pulse was 142; at 1.30 P. M. it was 140 and extremely arhythmic; at 2 o'clock it was 160. At 2.30 the temperature vaginally had risen to 108.2° F. and pulse to 160. At 3 o'clock she died, the last recorded pulse being 160, the respiratory rate having fallen to 40.

The diagnosis in a case of this sort at the time of the present prevalence of encephalitis lethargica might well have been so made. There was, however, about her evident signs of a severe meningitic process as well as a basilary one. One could not help but remember the caution of Pierre Marie, that the differential diagnosis between encephalitis lethargica and meningoencephalitis luetica cannot be made except by an examination of the cerebrospinal fluid.

The obstetric history was slightly suggestive in that the patient though married for some years had never been pregnant. Blood examination offered little information; the hemoglobin was 60, the red blood-cells 4,240,000, the white blood-cells 12,400. The differential count showed a polymorphonucleosis

of 81 per cent., with 12 per cent. of lymphocytes and 7 per cent. of large mononuclear cells. Such a cell count might very well have been found in an ordinary case of encephalitis lethargica.

When the patient was first seen by a physician, before she was brought to Mercy Hospital, it was thought that she might possibly be suffering from hysteria. Subsequently the hypothesis of a possible uremia was entertained, and inasmuch as albumin and a few casts were found, there was some disposition before her entrance into the hospital to insist upon this as a possible diagnosis. Three catheterized specimens, however, removed shortly after she reached the hospital showed no albumin, no casts, and no red blood-cells. One of the specimens removed March 29th showed acetone and diacetic acid, which were evidently due to the starvation consequent upon her inability to swallow and the rectal feeding which was made necessary.

Of course, a differential diagnosis between meningo-encephalitis luetica and encephalitis lethargica could only be made by the cerebrospinal fluid. Accordingly, very shortly after her entrance I did a spinal puncture. The fluid showed an increased globulin, an increase in cell count to about 60, and a 4+ Wassermann. Meanwhile the blood Wassermann had also turned out positive. Therefore the diagnosis made was meningo-encephalitis luetica. It was felt that the meningitic disturbances were very prominent, and that the brain stem, as shown by the behavior of the pontine and medullary nuclei, was also gravely affected. The course and the fever were altogether too spectacular for a diagnosis of gumma. Whatever the disturbance was, it was felt that it must be very acute and diffuse.

Postmortem examination was made the morning after her death. On examining the calvarium there was a high degree of cranial tabes. The left side of the skull showed rather deeper markings of the convolutions than the right. The pacchionian depressions were normal and the dura was normal. There was no increase in the intracranial cerebrospinal fluid. The brain was then removed within the dura. On examining the

base of the brain one was immediately struck with the condition
of the basilary artery. It contained a dark, formed clot about
4 mm. in length, organized firmly, and fastened to the walls
of the basilary artery so that it could not be removed. On
the basilary artery there was also a plaque which was thickened
and which was evidently the diseased area which had led to
the formation of the basilary thrombosis. This basilary throm-
bosis was undoubtedly the cause of the intense headache
which preceded the final cutting off of the basilary artery.
The brain itself showed no edema, and the meninges, both
dural and arachno-pial, were fairly normal. The circle of Willis
showed no changes, though the left internal carotid contained
a clot which was apparently not organized. The right inter-
nal carotid was perfectly free and open. There was marked
injection of the vessels all over the left side of the brain, which
may have been occasioned by the clot in the left internal carotid.
A very intense cerebritis was found also all over the brain.
The veins of the arachno-pia were engorged with dark blood.

Going back to the clinical symptoms, the correlation between
them and thrombosis of the basilary artery is very simple.
There is, first, the very intense disturbance of the pyramidal
tracts, such as would be occasioned by an irritative lesion, due
to the thrombosis of the basilary artery and the cutting off
of the blood-supply. At first the phenomena were irritative,
with patellar and ankle-clonus. After two days the effect
of the thrombosis was paralytic rather than irritative, with
the result that the patellar and ankle-clonus disappeared,
leaving only a paraplegia and the bilateral Babinski reflex.
Interesting also is the behavior of the respiration during her
illness. There was at first quite an interplay between the
paralytic and irritative effects upon the respiratory center.
At times the respiration would be extremely labored; at other
times it was quite easy. Moreover, the rate would vary un-
accountably, sometimes being very rapid and within an appar-
ently short time falling; thus, at 10 p. m. on March 26th the
respiratory rate was 52 and at 1 a. m. it was 34. Again, at
10.15 a. m. March 27th it was 44 and at noon it was 32.

Interesting also is the behavior of the pneumogastric inhibitory nucleus. For a long time this was the center of a good deal of irritation, with the result that the pulse was progressively slow, so that with a temperature of 104° F. a pulse as low as 76 was recorded. Later on, as this center became paralyzed instead of merely irritated, the pulse rapidly began to increase, so that at the time of her death it rose to 160.

Very easily to be explained, moreover, are the signs of pontine disturbance. We do no wonder that in the presence of a basilary thrombosis there should be trouble with swallowing and with articulation, and neither is it strange that there was a facial paralysis on one side. The wonder is that there was not a bilateral paralysis. It is, of course, perfectly evident that both sixth nerves must have been involved by such a lesion, and hence the bilateral internal strabismus is easily explained. The disturbance of the pupillary reflex is undoubtedly due to irritation of the fasciculus longitudinalis posterior, and is easily accounted for. In fact, all the symptomatology which this patient showed is referable to the zone of the pons and medulla and to the blood-supply of the basilary artery.

Had the patient not been unconscious we would have doubtless found many more interesting phenomena. The fillet must have been bilaterally affected, but we had no means of checking up on this apparatus. Undoubtedly, if we were able to make the necessary tests we might have found disturbances of equilibration, disturbances of both the auditory and vestibular apparatuses, and marked sensory disturbances in the territory of supply of the fifth nerve. Her unconscious state precluded the making of any of these observations.

This case illustrates beautifully the danger there is in looking upon acute cases involving the brain stem as uniformly examples of encephalitis lethargica. This is an error into which a clinician might easily fall, but if one is mindful of the fundamental fact that luetic meningo-encephalitis cannot be distinguished from encephalitis lethargica except by the Wassermann test and examination of the cerebrospinal fluid, he will not fall into such errors of diagnosis. As a final deduction

from this case I would suggest that bilateral intense irritation of the pyramidal tracts with evidence of pontine and medullary nuclear palsies, succeeded rapidly by abolition of ankle- and patellar clonus, but with preservation of the Babinski toe-signs, and evidence of marked irritation and then paralysis of the pneumogastric inhibitory nucleus (nucleus ambiguus) are the cardinal signs of basilar thrombosis.

VISCERAL SYPHILIS

THE patient entered the hospital March 30, 1921, having been referred to me for diagnosis and treatment by her physician, who sent the following history:

"Mrs. X., fifty years of age, married some twenty years. She is a nullipara, weighing 175 pounds or more, who was taken sick in August, 1920 with what her doctor called 'summer influenza.' He states that her sickness at that time was mildly febrile, with some general tenderness over the abdomen, with some looseness of the bowels and cough. I saw her October 17th and there was then marked looseness of the bowels and from time to time symptoms that varied from diarrhea to those associated with pulmonary signs with cough. Throughout there was rarely fever, but there was a continued and rapid loss of weight.

"Previous to the onset of the present illness over a period of several years there have been occasional attacks of pain in the gall-bladder area that were believed to be due to the presence of a biliary tract infection; at such times she has been advised to resort to surgery. During her present illness she has had no attacks of gall-bladder disease and, indeed, none for a long time preceding her present illness. Throughout her sickness there has been a recognized cardiac murmur, and during her early illness much stress was laid upon the abnormally 'hard beating' of the heart; and during this early period her pulse-rate varied from 90 to 100. In a degree these symptoms have subsided.

"Urinalysis did not seem to explain the continued loss of body weight, neither could any neoplasm or evidence of malignancy be discovered. The blood Wassermann test was negative, as well as all history of venereal disease. Her condition at first was thought to be in part due to cardiorenal changes,

but later the associate physician thought that we ought to operate upon her gall-bladder.

"Some seven years ago she had an unusual skin affection, which was diagnosed as pemphigus, and for which she was in the hospital for a month or more. This eruption was characterized, as I recall, by large bleb-like open lesions covering large areas of the extremities and of the body. Since that time she has been in fair health. I will enclose a few of the earlier laboratory reports, the later ones having been retained at the hospital where she has been for many weeks.

"We have been at a loss to arrive at any definite diagnosis, though syphilis has often been thought to be the most feasible. With this thought in mind cacodylate of soda was given for a long time without apparent effect, except that after some weeks she began to develop little indurations at each point of injection, which ultimately broke down and were opened. These discharged a clear and then cloudy serum, and ultimately they became black, dry, necrotic spots which loosened or were removed, leaving the open places now to be seen. The tissue heals very sluggishly. The patient is now much wasted, anemic, and of low vitality. She expects to go to Chicago Tuesday night and will go direct to Mercy Hospital."

The laboratory reports enclosed were as follows:

The urine on October 17th showed a specific gravity of 1020, acid reaction, the slightest possible trace of albumin, no sugar, a few hyaline casts, and a moderate amount of pus-cells. On the next day the twenty-four-hour specimen showed a specific gravity of 1024, the slightest possible trace of albumin, no sugar, a few hyaline and rarely granular casts, a few red blood-cells, many crystals of calcium oxalate, and some pus-cells. The next urinalysis was made on December 6th and was practically a duplicate of the preceding, with the exception of the specific gravity, which was 1016. A few hyaline and rare granular casts were present on that occasion. The last analysis of the urine was made March 22d. The specific gravity was 1031, reaction acid; there was no albumin or sugar; a few hyaline casts and a few red blood-cells were found, but no crystals.

Three blood examinations accompanied the patient's history, the first of these being made on October 18, 1920. For convenience they are put in tabular form:

Date.	Hemoglobin.	Red blood-cells.	White cells.	Polymorpho-nuclears, per cent.	Small lymphocytes.	Large mono-nuclears.	Eosinophils.	Basophils.
10/18/20	75	3,536,000	9,400	71	21	7	1	0
12/14/20	75	3,488,000	12,000	80	13	6	1	0
1/8/21	70	3,528,000	11,400	83	10	6	0	1

On the patient's entry into the hospital the history which she gave in the main coincided accurately with that which accompanied her. She amplified slightly by saying that during August she contracted a bad cold in the head followed by hoarseness. This cold left her extremely weak and shaky; even at the present time she still complains of a sense of tremor when she attempts to walk. Immediately following the cold she says that her heart went bad and that it has required medicine ever since. About this time she states that an epidemic of diarrhea set in in the town in which she lived, and that she acquired this diarrhea, which the doctor said was "summer influenza." This diarrhea has persisted ever since, being made worse on the slightest provocation.

The disturbance on the part of the heart began with swelling of the feet at night. During the night the swelling would disappear and when she got up in the morning she would not have any. Associated with the swelling of the feet and ankles, which is not so clearly cardiac in origin, she was also troubled by shortness of breath, palpitation of the heart, and general weakness. After the persistence of the cardiac disturbance for some time she began to be bothered with nocturia. She would rise four or five times at night and void rather large quantities of urine. Her family history, which is not mentioned in the history sent by her physician, discloses the fact that her father died of carcinoma at the age of sixty-six. Aside from

this there is nothing which in any way could have a bearing upon her condition.

A review of the history as sent by her physician states that she is a nullipara. We found on further questioning that she has been pregnant three times. She has had two miscarriages and a living child that died immediately at the time of birth. We have been unable to find out why this child died. The patient passed the menopause three years ago without trouble at the age of forty-seven.

Another point in her history which requires explanation because it proved of great value in the matter of diagnosis was a careful inquiry into her story of pemphigus. We learned that she had apparently a pemphigoid syphilis. She states that the surface of her body broke out in blisters rather widely distributed, and that for a month she was extremely ill. Nobody in her home town was able to do anything to relieve her condition, until finally a physician suggested that, being at the end of his resources, he was going to try a remedy which might be of value. She describes the remedy as "new blood" which was put into her vein. The "new blood" consisted of about a pint of lemon-yellow fluid. When asked if she remembered the name of the drug she promptly said that it was salvarsan. She further stated that with this injection and one or two others she was rapidly cured of the pemphigus and was as well as ever. Though her weight is stated in the history as having been 175 pounds when she was taken sick, as a matter of fact she weighed 216 pounds. She was able to give us definite information in this regard. Her present weight is 124 pounds, so that she has lost over 92 pounds since August.

Present Condition and Examination.—On entrance into the hospital her condition was rather deplorable. Her face was very much emaciated due to her great loss of weight, and its color was a peculiar one, suggestive both of an anemia and the bronzed subicterus of pancreatic disease. It was not lemon-yellow enough for pernicious anemia; it was not icteric enough for jaundice; it was not bronze enough for pancreatitis. She did not look cachectic enough to suggest carcinoma, notwith-

standing her great loss of weight. The arteries of the neck were pulsating violently and there was visible pulsation in the suprasternal notch. Her heart was evidently dilated. the apex-beat diffuse. Her pulse was not very rapid, being 106 at entrance, rising to 120 at night. reaching 134 on April 1st, two days after her entrance. This was the most rapid pulse-rate that she has had since her stay in the hospital. Associated with this rapidity of pulse was a great deal of inequality which was evident when her blood-pressure was taken. The beats varied in strength in millimeters of mercury some 10 or 15 points, the highest ones reaching a level of 160 mm. The diastolic pressure was also unequal. varying from 100 to 110. Besides the inequality there was a great deal of arhythmia. so that a notation appears upon her chart calling attention to the inequality and arhythmia of the pulse. Besides the bad heart action respiration was exaggerated. the respiratory rate being as high as 48 on some occasions and as low as 16 on others.

She also was running a febrile temperature, varying from 98.6° to 102.4° F. This fever persisted without very much change for a long period of time; indeed, it was not until May 1st that her temperature became permanently normal. This temperature seemed first to be due to numerous boils and abscesses which she had over the surface of her body. She had been given cacodylate of soda hypodermically, and at many of the points of injection. owing to the cachectic state of her tissue, the tissue had broken down and produced abscesses. These abscesses had apparently served as foci of distribution for further furuncles. so that numerous fluctuating areas over the arms. thighs. and buttocks had to be lanced. When these were lanced instead of frank pus appearing they seemed to contain some mucoid material and serum. They were very suggestive of iodin poisoning. though there was nothing in her history to show that she was taking iodin when she came to us. Indeed. she said that for a long period of time she had been getting no medicine whatever. These boils ultimately ceased coming. and now she is entirely free from them. though some thirty of them were lanced from time to time. While she had

them they were extremely painful and constituted her chief source of complaint. Indeed, she used to say that she could understand Job's feelings during the time that he was suffering from the plague of the boils.

Another source of discomfort to her was the evidence of broken compensation on the part of the heart. She was very short of breath, as has been stated, especially at times, but she also had swelling of both feet and ankles and a small amount of ascites. Her liver was not enlarged, but there was some tenderness beneath the right costal border. Her spleen·on entrance was not palpable, nor has it ever been palpable since. Examination of the heart showed, as has been stated, that it was dilated, but more important than the dilatation was the presence of a rather loud aortic systolic murmur with an accentuated aortic second sound. Such a murmur in an individual fifty years of age who has not previously suffered from a rheumatic infection of the heart should raise the presumption of syphilitic aortitis. An x-ray was made of the chest and showed a dilated aortic arch. There was no true aneurysm, but merely a diffuse dilatation. In the presence of her obstetric history, the story of pemphigus which had been healed by salvarsan, the onset of a cardiac malady at the age of fifty without an antecedent history of rheumatism, the diagnosis of aortitis was most certain, and notwithstanding the negative Wassermann, which was reported in her history, another one was accordingly made. This gave a 3+ reaction, which was regarded as being of quite as much significance in view of her history as if it were frankly 4+.

Another source of complaint was her stomach and bowels. These caused her a great deal of trouble. On April 1st she vomited about 10 ounces of a light green liquid. The report from the laboratory showed that it contained no blood and no free hydrochloric acid. After this first attack of vomiting she remained free from emesis until April 6th, when she again vomited about 6 ounces of a bile-stained fluid. She complained a good deal about heart-burn; though numerous attacks of nausea succeeded this vomiting for a time, it was not until the

27th of April that she again vomited. On this date she vomited on three different occasions. A Rehfuss analysis of the gastric contents showed no free hydrochloric acid and the total acidity varied from 6 to 24 in seven different specimens collected at half-hour intervals. Bile was present in large quantities in all seven specimens. The diarrhea which she had during last August occasionally reappeared, and was attributed to an achylia gastrica which was found on the Rehfuss test.

Several blood examinations were made on March 31st, April 4th and 18th, and May 12th and 17th. In general, these blood examinations are not much different from those which were made before the patient entered the hospital except in the matter of the percentage of hemoglobin. The percentage of hemoglobin as determined in this hospital has varied from 52 to 68 per cent. and the red blood-cells from 3,200,000 to 3,410.000. Two blood-cultures were made on the 3d and 23d of April. They were both entirely negative. Urinalyses were made March 31st, April 1st, 5th, 9th, 30th, and May 13th. The last urinalysis was made May 13th. It was a twenty-four-hour specimen and consisted of 700 c.c., with a specific gravity of 1026, acid reaction, with no albumin or sugar, and no casts or red blood-cells.

Diagnosis.—The diagnosis in this case was suspected because of the cardiac findings. On physical examination the first thing to strike one's attention was the aortic systolic murmur. An aortic systolic murmur coming on in a patient about fifty years of age without previous cardiac disease should raise the presumption of syphilis. It therefore raised such a presumption in us, and we immediately began cross-examining her on the history. The first point was the discovery that she was not a nullipara, but that she had had two miscarriages and a still-born baby. The next discovery was that she did not have pemphigus, which is a fatal disease, but, on the contrary, pemphigoid syphilis, and that this was cured by salvarsan. We then found by further physical examination that the patient had signs of arterial degeneration everywhere. We were confronted with the fact that she had a long history of apparent gall-bladder

disturbance, together with a history of very marked loss of
weight. Such an extraordinary loss of weight as she shows
when not due to malignant disease or suppuration is apt to be
due to pancreatic disease. The diarrhea could not be held
accountable for it, for it had not been sufficiently intense.
Such a diarrhea as she had is very properly to be ascribed
to the achlorhydria. Moreover, the achlorhydria needs some
explanation. What could be the etiologic factor? The whole
problem seems to resolve itself into the question of what visceral
lues can do to a patient. Ordinarily, lues of the big abdominal
viscera, notably of the pancreas, to some extent of the liver,
and as far as we know also to some extent of the stomach,
is due primarily to an arterial disease of the blood-supply of
these various organs. Is it not a fair supposition that she is
suffering from luetic endarteritis of the celiac axis? Can we
not explain the apparent history of gall-bladder disturbance
which runs through the past, the present disturbance on the
part of the stomach, and the excessive loss of weight on the
basis of a luetic endarteritis following the arteries into the liver,
stomach, and pancreas? Is not the anemia possibly in some
way bound up with a syphilitic arterial disease of the spleen?

Since her stay in the hospital the ascites of which she had
a very slight amount increased materially, so that early in May
it was necessary to tap her abdomen; 8 quarts of ascitic fluid
were removed. Instead of getting the usual straw-colored
fluid which one would expect, we were surprised to find a milky
fluid exuding. The question immediately arose whether it was
a chylous or chyliform fluid, and we have had much difficulty
in trying to answer this question. A chylous ascites tends to
accumulate very rapidly, so that large volumes have to be
removed repeatedly. In her case we have been obliged to
tap but once; although she has reaccumulated a certain amount
of fluid since May 9th, still at the present time it is not sufficient
to justify another tapping.[1]   Chylous ascites is usually yellowish-

[1] Subsequently the patient was tapped for diagnostic purposes, and 4
quarts of a *straw-colored* fluid was removed with no trace of chylous fluid.
Examination showed it to be an exudate.

white in color, whereas this fluid was more of a milky white and seemed a most perfect emulsion. Chylous ascites occasionally has an odor suggestive of fat, but this fluid was without any odor. Chylous ascites sometimes acts like milk in that a creamy layer may form on its surface when it stands, but in the case of this fluid no creamy layer separated out. These points would lead one to suspect that the fluid which we found was chyliform rather than chylous, yet the laboratory work done upon the fluid showed a specific gravity of 1018, while chyliform fluid usually has a specific gravity of 1012 or lower. On account of this high specific gravity, 1018, instead of looking upon this fluid as ordinary chyliform transudate, it must be looked upon as chyliform exudate. The amount of protein was determined in the fluid and was found to be 1.2 per cent. or 12 grams per 1000. This was an amount of protein which is more like that found in chyliform fluid rather than chylous. In chylous fluid the protein does not usually exceed 3 grams per 1000, whereas this fluid showed 1.2 per cent. Moreover, this fluid showed red blood-cells and gave a positive benzidin test for blood. Chylous fluid ought not to contain any blood.

One might infer from the presence of blood-cells that the fluid was carcinomatous in origin, that the chyliform ascites was due to some peculiar change taking place in it after it had been collected in the body cavity. It is, however, possible to get such chyliform ascites in gummata occurring in the abdominal cavity upon a peritonitic surface. When syphilitic peritonitis arises over the surface of the gumma, in other words, when peritonitis has been started in this manner, it is possible for the infection to spread over the peritoneum in general so that chyliform ascitic fluid may develop as an exudate. We are of the opinion that the ascites in this case is of the chyliform type and that it is further evidence of abdominal lues.

Treatment.—The patient came to the hospital with a temperature ranging as high as 102.4° F., with respiration averaging 48, and with a pulse as high as 134. She weighed 124 pounds and her heart was beating wildly. She had numerous boils which were very painful. Her abdomen was distended, she

was so weak that she could scarcely move in bed, and it seemed as though nothing could be done for her, and yet, believing as we did that her trouble was luetic in origin, she was given her first injection of 0.45 gram of neo-arsphenamin. Since her stay in the hospital she has had three fractional and three full doses of neo-arsphenamin. She has been without any fever since the first of May. She is now out of bed the major part of the whole day. She sleeps well, feels well, and seems to be in every way recovering. Aside from the neo-arsphenamin the only other treatment which she has had was a small amount of digifolin, 3 tablets a day at first until her heart gradually became perfectly under control, and a small amount of iodalbin, namely, 5 grains four times a day. She has also been given of late 5 grains of pancreatinin three times a day, which may or may not be benefiting her; we are unable to state. We do, however, feel positive that the great amount of improvement which she has had is due to the injections of neo-arsphenamin. The following notations from her history sheet will better convey her present condition:

May 16th: Sitting in chair; feeling fine; up and about; good day; sleeping.

May 17th: Slept seven hours; sponge bath; feeling fine; good day.

May 18th: Slept seven hours; sponge bath; up and about; feeling better today; good day.

May 19th: Slept seven hours; up for one hour; feeling fine; up all day; complains of feeling tired; sleeping.

Indeed, her present state is practically one of convalescence. We shall not cease our antiluetic treatment; we have persisted in it because we believe that it is still doing her good. We have not much confidence in ultimately completely curing visceral lues, because we believe that a large amount of visceral lues is dependent on arterial syphilis, and such arterial syphilis is very hard to control. We do believe, however, that it can be controlled to some extent, and in so far as it can be, the general condition of the patient can be much helped. We see no reason to change our primary conception of syphilis

of the celiac axis. We cannot look into her abdomen and see all the pathologic changes which are taking place there, but if we had the opportunity we should certainly spend most of our time in making a careful microscopic examination of the arterial trunks. I am of the opinion that most obscure visceral lues, of which this is an undoubted example, are really arterial in origin. I believe that the inflammation affecting the arterial trunks is responsible for a good deal of the unexplained pancreatitis and the obscure functional derangements of liver, stomach, and spleen.

I cannot but believe that the blood-stream in this patient's case is also affected by the syphilitic infection, and in this connection we have omitted one extremely interesting finding. Just as the boils were ceasing to appear a bleb, the only one upon her body, appeared on the cheek just beneath the left eye. This grew rapidly in size until it was nearly ¾ inch in diameter. We dressed it with boric acid saturated solution and left it to itself. It healed as did the rest of the disturbance upon her body, but following this healing we made a rather remarkable discovery in her blood. The differential count, which had shown up to that time a normal number of polymorphonuclear cells, suddenly changed. From 72 per cent. the polymorphonuclears dropped to 48 per cent., the small lymphocytes dropped from 25 to 23, the large mononuclears dropped from 3 to 1 per cent., and polymorphonuclear eosinophils suddenly appeared in the blood to the extent of 28 per cent. We could scarcely credit this change, though it was attested to by several competent workers. Another examination made a few days later still showed eosinophils 26 per cent. This finding, which is rather unique and puzzling, may perhaps be correlated with the bleb beneath the left eye. It is possible that the eosinophilia was due to a mild recrudescence of the pemphagoid lues, of which the only cutaneous manifestation was the one spot upon the face. Eosinophilia in general when slight is of very little diagnostic significance. The conditions in which eosinophilia is marked are found practically only in various parasitic infections and in certain skin diseases, particularly those in which bullæ

form. She shows no parasitic disease of any sort. There is no trichinosis about her; she has no tapeworm; she has no hookworm, and it would seem as though in her case the only explanation for this eosinophilia must be the dermatosis, the only other manifestation of which was the cutaneous eruption on her left cheek.

This case is presented more for its suggestive diagnostic points than for any demonstration of pathology. It is evidently an obscure visceral lues with chyliform ascites which is difficult to account for, with a rather strange eosinophilia, and yet with remarkable improvement on the use of antiluetic measures. It is probable if we could have a very careful postmortem examination at some future date that it would disclose to us how visceral lues comes about. We are positive of one thing, namely, a syphilitic hepatitis. It is not unreasonable to suppose that the remainder of the visceral disturbances are in the last analysis arterial in origin, and surely if the celiac axis is the particular artery involved it is not hard to explain the whole case on a comparatively simple pathologic hypothesis.

CLINIC OF DR. ISAAC A. ABT

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MENINGEAL HEMORRHAGE OF NEWBORN AND YOUNG INFANTS

THIS morning I desire to call your attention to a little patient who is but a few days old and who presents a condition which is not at all uncommon among newborn infants. It is frequently the cause of death among the newborn and very often causes mental and motor deterioration, retarding the patient through his entire life, and frequently rendering him a helpless paralytic and a hopeless idiot or epileptic.

This baby is ten days old. He was born of a primiparous mother. The labor was not protracted and a low forceps delivery was employed without using force or pressure to complete labor. The child's color was good at birth, but very soon he became cyanotic. The fontanel has been protuberant, the pupils are small, and he has presented a slight nystagmus. The baby takes the breast very poorly and does not seem at all hungry. He is difficult to arouse, and upon examining him further you will notice that his extremities tend to be rigid, that his pupils react slowly if at all, and that his breathing is rapid and superficial. He has had a slight temperature varying between 100.5° and 101° F. You will notice also that when he is aroused he gives a short, penetrating cry which does not resemble that of a normal newborn infant. There can be no doubt that this baby is suffering from meningeal hemorrhage, which will satisfactorily explain all the symptoms here presented.

The intracranial hemorrhages of the newborn and young infants are of more frequent occurrence and of greater impor-

tance than is generally considered. The conditions under which the hemorrhage occurs, the most frequent locations, and general pathology are not at all clear. However, to one who reviews the literature and considers his own experience there is presented an unusually interesting and important clinical condition.

Intracranial hemorrhages of newborn infants are not infrequent. According to the statistics of the pathologic institute at Kiel, Heller found 1014 cases in 5998 autopsies on children. In other words, 17 per cent. showed intracranial hemorrhages or at least traces of hemorrhages. Of the above number of cases, 307 were born dead; 249 died during the first two weeks of life; 252 during the first quarter; 161 during the remaining three-quarters of the year; and in the second year there were only 52.

The hemorrhages occur under a variety of conditions. Long-continued and difficult labor, especially in primiparous women where a disproportion exists between the size of the child's head and that of the birth canal, is one of the most frequent causes. On the other hand, meningeal hemorrhage occurs sometimes in precipitate labors and labors which appear to be normal. But difficult labors of any type are more apt to be attended with hemorrhage.

Application of the forceps has not been without its toll of hemorrhage. This operation increases the overlapping of the parietal bones. Sometimes the forceps produce funnel or spoon-shaped depressions in skull bones. Occasionally the pressure is so extreme and the hemorrhage so profuse that the infants die during birth or come into the world in a dying condition. In some instances, however, the depressions due to forceps do not seem to have any influence on the brain or its membranes during the first period of life. Careful statistics have shown that in 50 per cent. of the cases the brain was uninfluenced by the depressions produced by the forceps, but one must not be unmindful of the fact that later complications, particularly epilepsy, may have had their origin in the trauma produced by the forceps.

Hofmeyer observed a series of 25 cases in the Würzburg Clinic, in all of which the infants had been delivered by forceps;

7 died during birth or some hours afterward, and in all of these cases autopsy showed bone injuries and intracranial hemorrhages. Of the remaining 18 cases, 5 died during the first year from intracranial diseases, and 12 of the others were observed for a considerable period. Notwithstanding the compressions produced by the forceps, the infants were able to develop normally as far as physical and mental health were concerned. Hofmeyer concludes that compression of the skull of newborn infants does not always produce the dire results which are so frequently expressed.

It has been estimated 'that 30 to 40 per cent. of infants delivered by forceps suffer intracranial hemorrhage. In fact. Wilcox states that in a choice between a protracted forceps delivery and a cesarean section the balance of safety to mother and child is enhanced by resort to the latter. As opposed to this view Kustner reports a case in which the moment pains began a cesarean section was performed. Nevertheless, hemorrhage into the meninges took place. Some students have considered this bleeding the result of skull compression following the incision of the uterus, and state that Kustner's case is not one of intra-uterine hemorrhage.

Severe trauma of the skull produced by high forceps operations or long-continued pressure of the forceps often leads to fissures or fractures of the vault, and sometimes fractures of the base and tearing of the sutures. The hemorrhage which occurs in these cases is usually of the severer type.

Among other operations whose severity causes sufficient trauma to produce hemorrhage is the Smellie-Veit maneuver and podalic extraction. Runge found that rupture of the vertebral column had occurred in 64 cases of the latter condition, with death of the child. There was a separation of the intervertebral cartilages from the body of the vertebra.

We should again recall that most investigators agree that intracranial hemorrhages in infants may occur without manifest injury to the cranium, and that even easy labors with spontaneous delivery may result in hemorrhage.

Breech presentations are frequently attended with men-

ingeal hemorrhage. In these cases it may be assumed that the maternal parts have not been given sufficient time to undergo dilatation. Consequently, there is prolonged compression of the head and an overriding of the skull bones.

Prematurely born babies are particularly disposed to meningeal hemorrhage. In fact, premature birth should be considered a traumatic process in which characteristic pathologic conditions are found. Of these conditions, intracranial hemorrhages are the most important. Yippo believes that they are responsible for 30 per cent. of the deaths of prematures in the early days of life; 18 per cent. of these easily and spontaneously born show intracranial hemorrhages.

Asphyxia is an important factor in meningeal hemorrhage. There has been considerable discussion whether the asphyxia or the hemorrhage appears first. As a matter of fact, the process may work both ways, and in any particular case it may be difficult to determine which was cause and which was effect. A large clot may produce pressure symptoms, with injury to the respiratory center, and asphyxia may result. On the other hand, asphyxia produces congestion, and hemorrhage may result. It has frequently been observed that the cases of asphyxia also show hemorrhages into the pleura, pericardium, and thymus gland. Asphyxiation tends to increase the flow of blood. Incidentally, it might be added that asphyxia occurs in connection with congenital syphilis. In 122 cases of asphyxia Weyhe found 23 cases of congenital lues. It also occurs in breech presentations with difficult delivery of the after-coming head, in congenital goiter, and in cases where the cord is tightly wound around the neck.

The hemorrhagic diatheses of the newborn are frequently associated with hemorrhages into the spinal canal and meninges.

The vessel walls, themselves, are sometimes at fault. This is particularly so in luetic subjects and in prematures, whose vessels have not attained their full development.

In addition to what one might term the accidents of labor there is a certain group of cases due to bad obstetric practice. How frequently we find in taking the birth history of our cases

of Little's disease that the intern or nurse has held back the head and prevented a spontaneous birth of the child in order to give the accoucheur the opportunity of conducting the birth.

Pathology.—The most frequent condition giving rise to meningeal hemorrhage is the overlapping of the cranial bones during birth. As a consequence of this overlapping the sinuses lying under the sutures and particularly the veins which empty into the sinuses become compressed. The veins are pulled, and eventually the walls are torn. This is more likely to occur in severe asphyxia resulting from general venous congestion. The hemorrhage may not only occur from laceration of the veins, but the force applied may be sufficient to cause a tearing of the dura itself. These occur mostly in the tentorium and are usually confined to the upper lamellæ.

The so-called subdural hemorrhages and tears of the tentorium which are present in full-term infants are only exceptionally seen in prematures.

The arachnoid hemorrhages arise from tearing of the veins in its network, and the effusion as a rule is sufficient to lift the arachnoid from the pia. The hemorrhages into the arachnoid, the ventricles, and the brain substance itself are usually due to compression of the skull at birth.

In general, one frequently finds small fresh hemorrhages in the region of the longitudinal sinus over the hemispheres. They may be located over certain definite convolutions, but there does not seem to be any place where they occur with any particular predilection. The larger hemorrhages gravitate downward and are found at the base.

Ventricular hemorrhages are seen particularly in stillborn children, though they are found up to the second year. In the majority of cases they are due to a rupture of the choroid plexus, although some result from small hemorrhages of the ependyma. The lateral ventricles are most involved, and the bleeding has been thought to be the result of marked compression or displacement of the skull bones in the region of the lambdoidal suture. As a result of this overlapping of sutures the great veins of Galen become compressed and pulled. Con-

sequently, there is a congestion of the choroid plexus followed by extravasation of blood. Ventricular hemorrhages are frequently found in premature infants.

Hemorrhages into the brain substance itself may occur, though they are rare.

Infection may occur as a result of bacterial invasion of the foci of hemorrhage.

Seitz has classified meningeal hemorrhage according to the clinical and pathologic aspects, into the supratentorial and infratentorial types. His classification is the one most widely accepted. The supratentorial are mostly unilateral. The hemorrhage covers the brain in flat layers, compressing the underlying cerebral cortex. Extension of blood to the other side of the brain, the infratentorial space, or the spinal canal is not frequent. The increased intracranial pressure prevents extensive spreading of the blood.

Seitz explains that the veins emptying into the superior sagittal sinus are distorted, but rupture of the sinus itself is rare. The majority of autopsies show that the hemorrhage occurs in the parietal and frontal regions close to the longitudinal sinus. Those hemorrhages are usually bilateral. Next in frequency is the vicinity of the tentorium. In this case overlapping of the occipital bone causes laceration of the tentorium. These hemorrhages are most fatal.

As has been mentioned before, hemorrhages arising from the veins opening into the longitudinal sinus are brought about by the overlapping of the parietal bones. The overlapping produces such pressure that the veins are not able to empty themselves into the sinus and are lacerated and torn away. Tearing of the sinus itself is rare, and if it occurs fatal hemorrhage is inevitable. Infratentorial hemorrhages which surround the cerebellum arise from the veins of the transverse sinus. Here again the sinus is not torn. The blood pours out into the spinal canal, although it does not seem to be forced upward into the tentorium itself.

It is to be noted that babies with small, soft skulls are predisposed to meningeal hemorrhage. In these cases the tearing

of the soft parts of the skull occurs very easily because the bones tend to overlap more readily than in well-developed babies.

Benecke has called attention to the frequency with which the tentorium is torn. He found this injury 14 times in 100 autopsies on newborn babies. If the infant's skull is pressed from side to side by the birth pressure the longitudinal axis of the skull becomes increased. Consequently, the tentorium is torn at right angles to the longitudinal axis. The lower leaf of the tentorium, the one corresponding to the cerebellum, is less frequently injured. Where the cerebellar portion of the tentorium is torn the hemorrhage covers the base of the occipital and temporal bones and sometimes the surface of the cerebellum.

The clinical classification of Seitz describes the supratentorial type of hemorrhage, in which focal symptoms referable to the motor areas are produced, and the infratentorial variety, in which bulbar and spinal symptoms occur early. This inferior type offers a less favorable prognosis.

Subarachnoid hemorrhages are usually small, flat, and multiple. They involve both hemispheres and usually depend upon congestion with or without asphyxia. As a rule they exist without producing clinical symptoms.

Infracerebral hemorrhages, in so far as they are associated with skull fractures, usually affect a small area of brain tissue.

It is a noteworthy fact that small hemorrhages into the meninges, punctiform in character and profusely scattered over the convexity of the skull, are commonly observed in autopsies on young babies. Weyhe found small hemorrhages in 12 per cent. of cases which he autopsied, and Doehle found them in 14 per cent.

F. C. Rodda, of Minneapolis, in a paper read before the American Medical Association in 1920, has made a very valuable contribution to the subject of meningeal hemorrhage in infants. His studies in coagulation time throw a new light on the cause and nature of the hemorrhage. He found among other things that the average coagulation time in the normal newborn infant is seven minutes, though it may vary under normal conditions from five to nine minutes. He found also

that there is a prolongation of the coagulation and bleeding time up to the fifth day of life, though this time gradually tends to recede by the tenth day, after which there is a return to the average first day's determination. These findings coincide with the clinical evidence because meningeal hemorrhage frequently begins after the first day and tends to continue as a profuse bleeding or oozing up to the fifth day.

Rodda draws some very valuable conclusions. Among these may be mentioned the fact that cerebral hemorrhage is not always caused by obstetric operation; it may follow normal labor when least expected. Severe trauma results in massive hemorrhage and early death. Rodda points out that hemorrhage may occur as a result of the constitutional state, predisposing the infant to hemorrhage and, in addition, the possibility of a slight trauma. Babies with meningeal hemorrhage bleed slightly at first, though later on continuous oozing occurs, leading to the accumulation of a considerable quantity of blood.

If one detects a tendency toward bleeding or if the bleeding is gradually taking place, the patient should receive some hemostatic preparation, preferably human blood-serum, normal horse-serum, or one of the other hemostatic ferments.

Internal cephalohematomata are situated between the cranial bones of the dura. They occur as the result of injury to the bone, and very frequently correspond in situation to external hematoma, particularly when the latter are situated over the ossified portion of the parietal or occipital bones.

Hemorrhage into the spinal meninges may be located between the dura and the vertebræ or may occur between the dura and the arachnoid. While it may be primary in the spinal membrane, such occurrence is probably rare. This form is usually the result of skull fracture or extensive hemorrhage into the middle or posterior cerebral fossæ, with extravasation of blood into the spinal membrane. In a series of autopsies Littzman found that the presence of blood in the arachnoidal space was indicative of extensive hemorrhage, and the source of the blood was usually intracranial.

Symptomatology.—The symptoms will necessarily vary with

the size and location of the hemorrhage, and will also depend on whether one or several regions of the brain have been involved. The initial symptoms vary from stillbirth to no manifestations at all. Sometimes slight symptoms are present at birth which increase in severity as the extravasation continues. Occasionally infants improve after birth and only gradually do the symptoms develop. The symptoms may be delayed until the fourth or fifth day. A presumptive diagnosis can be made in any baby without the classical signs of meningeal hemorrhage when the infant becomes pale, refuses nourishment, and shows a peculiar facial edema a few days after birth.

Increase in the amount of extravasated blood leads to an increase of cranial pressure and compensation occurs by widening and protrusion of the sutures and fontanel.

Occasionally during the first days of life the children are restless, cry, and refuse to drink, but injury to the respiratory center may interfere with crying. When contusion of the brain has occurred the children are quiet. The pulse is frequently slow and the bradycardia increases, although the vague center is not so irritable in young infants.

On the other hand, the vasomotor center is stimulated by the increased effusion, and this is evidenced by strong pulse and accentuated secondary aortic tone. On account of the vasoconstriction there is marked pallor. During the convulsion the skin is flushed.

If there has been restlessness at the beginning, which gradually disappears, the child tends to become somnolent and eventually may pass into deep coma. He does not react to external stimuli and does not cry when changed or handled. The desire for food is lost, and strong children no longer take the breast, though the sucking reflex may be stimulated by placing the nipple in the mouth.

Convulsive seizures are the most diagnostic and characteristic symptoms of meningeal hemorrhage. The convulsions are tonic and clonic and are produced by the slightest external stimuli. The entire musculature may be involved, and sometimes the respiratory muscles participate in the convulsion.

Many transitional forms occur. They vary from slight twitchings, which hardly change the child's appearance, to severe epileptiform convulsions with tonic contractions of all the muscles. The convulsions are usually bilateral. They may be unilateral, however, and sometimes only a certain group of muscles is involved. In the latter case a focal lesion may be indicated. The facial nerve may show a paralysis on the side opposite the hemorrhage. Spasms of the arms, legs, or sternomastoid muscle of one entire side may occur, pointing to a hemorrhage on the opposite side. Sometimes the upper extremities are more involved than the lower. Trismus is not frequent. In the hemiplegic type spasms and paralytic symptoms may be overshadowed because similar changes occur on the other side of the brain following compression and edema of the other cerebral hemisphere.

The eyes are very frequently involved. Sometimes there is ptosis, strabismus, and myosis. Myosis and ptosis usually occur on the same side as the hemorrhage, although this is not constant.

Respiratory spasms may occur with marked cyanosis, and Cheyne-Stokes' breathing is often present.

As the hemorrhage progresses considerable elevation of temperature usually occurs.

Epistaxis and bleeding from the pharynx are frequently found.

Yawning and sighing are of frequent occurrence in the hemiplegic type.

Hypertonia is always marked. Both the skin and tendon reflexes are exaggerated and the Chvostek sign is sometimes positive. The irritation is usually followed by paralysis. Tendon and skin reflexes disappear and death occurs on the fourth to eighth day.

Frequently death is due to an aspiration-pneumonia. Certain observers have described pulmonary atelectasis in cases of meningeal hemorrhage.

The termination is not always fatal. In some cases the irritation passes and the convulsions decrease, sometimes dis-

appearing within a week. The child takes food and reacts to his environment. The turning-point seems to be the third or fourth day. If the symptoms increase after this, death is to be expected.

There are a few additional interesting symptoms which should be mentioned before leaving the consideration of the symptomatology. Protrusion of the fontanel is often absent in the beginning, and only occurs when in cases of anterior cerebral fossa hemorrhage consecutive congestion and edema occur. In extensive infratentorial hemorrhages death usually occurs. In hemorrhages into the posterior fossæ hours may elapse before dangerous symptoms appear. Hemorrhages into both cerebral fossæ usually cause death the first day. Hemorrhages into the lateral ventricles resemble the infratentorial variety and the convulsions may be confined to the face. Very often the picture of ventricular hemorrhage resembles tetanus. Hemorrhages of the fourth ventricle and the spinal canal produce medullary symptoms.

Complications and Sequelæ.—One is surprised in studying the pathology of newborn babies to find that suppurative meningitis is rather frequent. It is usually considered that this is due to an infection, either by continuity or through metastatic processes. Gustav Lindberg has examined 4 such cases in young infants. His studies led him to the conclusion that there was meningeal hemorrhage at birth. Following this, bacterial invasion occurred, which resulted in purulent meningitis. He believes that the infection enters the hemorrhagic area of the meninges through the blood channel. In one case the baby had an infected umbilical granuloma. One baby suffered from erysipelas. In the other 2 cases no focus could be determined. As a result of his observations he thinks that hemorrhagic effusions which occur frequently in young infancy offer a point of low resistance. This tends to become infected and consequently develops into pyogenic meningitis.

In the pathologic study of cases of intracranial hemorrhage one finds frequently enough concurrent lesions in the lungs under the pleura and scattered through the parenchyma of

the lung. Similarly, hemorrhages are observed into the adrenal bodies, the skin, and the pericardia.

It is unnecessary at this time to go into detail concerning the sequelae which occur during the later life of the baby who has suffered from meningeal hemorrhage. The entire chapter of infantile cerebral palsies revolves to a considerable degree around this etiologic moment. The hemiplegias, the diplegias, and the quadriplegias, with their symptoms of rigidity, contractures, tremors, atrophy, athetoid and choreiform movements, are so well known to every practitioner that one need not go into detail concerning this symptom group.

Retarded mental development or idiocy of every degree may result from the degenerative processes produced by the meningeal hemorrhage. Possibly the most frequently resulting condition is epilepsy, which occurs in two-thirds of all the cases. On the other hand, it must be stated again that in some cases meningeal hemorrhage of moderate degree may occur, especially small foci, without leaving any permanent defect in the individual.

The results of the hemorrhage on the life and the health are various. If the hemorrhage is profuse, if it involves the base of the brain, and if it occurs intrapartum the child is usually dead when born.

The prognosis depends somewhat upon the stage of the asphyxia at birth. If the asphysia is deep, the resuscitation is difficult, or the baby revives for only a few moments, quickly relapsing, the condition may be considered as very grave. As has already been noted, in some cases infection of the hemorrhagic area may occur and a pyogenic meningitis may ensue. The ultimate fate of the baby depends upon the location and extent of the hemorrhage and the degenerative changes in the nerve cell and fiber that follow. Results may vary from trivial paralysis to extreme idiocy and extensive paralysis.

Lumbar puncture assists materially in determining the diagnosis. In cases where the differential diagnosis is difficult the bloody fluid obtained by lumbar puncture points to a subarachnoid hemorrhage. It must not be forgotten that bloody

fluid may be obtained on account of a mechanical blunder in technic.

Treatment.—Various procedures have been recommended in the treatment of these infants. Possibly the most striking method suggested has been that recommended by Harvey Cushing. He reported several cases of operative interference. In 1910 he reported 12 cases, several of which were bad surgical risks, with extensive contusion of the brain associated with laceration of the closing membranes. About half of these patients died during or soon after the operation. Others recovered without developing spastic paraplegia. Cushing says that several of the patients have done remarkably well, although athetoid states have remained in a few, which he said were due to complicating extravasations in the basal ganglia. He states that although the surgical procedure is usually well borne, it is a delicate and very difficult operation to carry out, and until much more can be learned regarding the late results and the best methods of procedure he concludes that surgical interference cannot be widely advocated for general use.

His method, very briefly, consists of making a parietal opening, a horseshoe incision, and the fibrous tissue of the fontanel is sectioned. The dura mater is incised to permit the escape of blood-clots. The subarachnoid cavity is then washed with warm saline solution, closing the wound.

Fontanel puncture has been suggested by Gilliss instead of this extensive operation of Cushing's. This procedure is practically free from danger. It diminishes the compression and is said to reduce the danger of infection of the effused blood. It diminishes the chances of the formation of a large clot. It is less hazardous than the more extensive operation, and it aims to accomplish the same purpose by removing the collection of blood, and, consequently, avoiding the compression of the cerebral cortex and the meninges.

Lumbar puncture has also been suggested as a therapeutic measure, and while in some instances of basal hemorrhage it may give slight relief, it seems to be a less efficient procedure than the fontanel puncture.

After a consideration of the frequency of permanent injury following the application of forceps one might come to the conclusion that the forceps operation is a serious procedure. In this connection it is interesting to note that many clinicians believe if forceps were applied earlier by skilful hands in cases where pelvic disproportion exists there would be less compression of the skull and less danger of meningeal hemorrhage.

CLINIC OF DR. SOLOMON STROUSE

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DIETETIC THERAPEUTICS

Cases Showing What Can Be Done in the Way of Dietetics under Properly Controlled Institutional Care; Discussion of the Present Status of Certain Phases of Dietary Therapeutics in Diabetes. Illustrative Cases.

CASE I.—HYPERTENSION. THE EFFECT OF HIGH PROTEIN AND HIGH SALT INTAKE

A RUSSIAN Jewess, married, age forty-seven, who has been in the hospital frequently with attacks diagnosed as chronic interstitial nephritis and hypertension, entered on January 6, 1921, after six months' remission. She left the hospital the last time in June, and was in fairly good condition until two months ago, when she began to have pain in the extremities and neck, and muscular weakness, shortness of breath, palpitation, headaches, and constipation. On admission she presented an appearance such as you see now—a rather large, stout woman in whom the positive findings are: a heart enlarged, 13½ cm. to the left, a loud, blowing systolic murmur heard all over the heart, but loudest in the aortic area. Pulse was regular, 86 to the minute, and full, with high pressure. The abdomen was full. The liver was palpable four fingerbreadths below the costal margin. There was no ascites. Slight edema of the ankles was present. Varicose veins were found on both legs. Blood-pressure on admission to the hospital was 290+ systolic (the machine would not register any higher) and 160 diastolic.

A twenty-four-hour urine specimen showed specific gravity 1025, a trace of albumin, a few hyaline and granular casts.

The urinary findings were constant until the latter part of her stay, when both albumin and casts entirely disappeared.

During her admission to the hospital she had an attack of what looked like acute cholelithiasis.

The x-ray showed a diffuse enlargement of the heart and a dilatation of the aorta. The Mosenthal test showed a slight fixation of the specific gravity. The night urine was 360 c.c. as compared to 370 c.c. during the day, total nitrogen excretion of 10 grams, total chlorids of 7, and total urea of 6.7.

Blood: Non-protein nitrogen was 32 mg., urea nitrogen 20.5, blood-sugar 0.121. The Wassermann test was negative.

In brief, this patient has a chronic hypertensive cardio-vascular disease which probably did not originate in the kidneys, but which probably belongs to the group known as essential hypertension.

The patient was immediately put to bed and the usual treatment instituted, that is, absolute bed rest, a salt-free non-nitrogenous diet, mild sweats, saline catharsis. As can be seen from the accompanying chart (Fig. 56) after four days of this régime the blood-pressure dropped to 260/150, and remained at that level for six days. The patient felt no better, and purely from an experimental standpoint we decided to see what the addition of protein and salt would do. No disturbance in the protein and salt metabolism had been proved, so we added to the diet meat, making a total protein intake of 75 grams, and 10 grams of sodium chlorid. The chart again illustrates how quickly and steadily the blood-pressure dropped immediately after this régime was started until ten days afterward, when the pressure was down to 200/100.

On the ninth day she developed intense abdominal pain, which, curiously enough, did not increase the blood-pressure, but because of this pain and her general distress associated with it she was again put back on a milk, toast, and cereal diet. From this time on her general condition remained constant, and despite all treatment she complained constantly of headache and general distress. Furthermore, despite the change of diet to milk, toast, and cereals, and finally to a salt-free non-
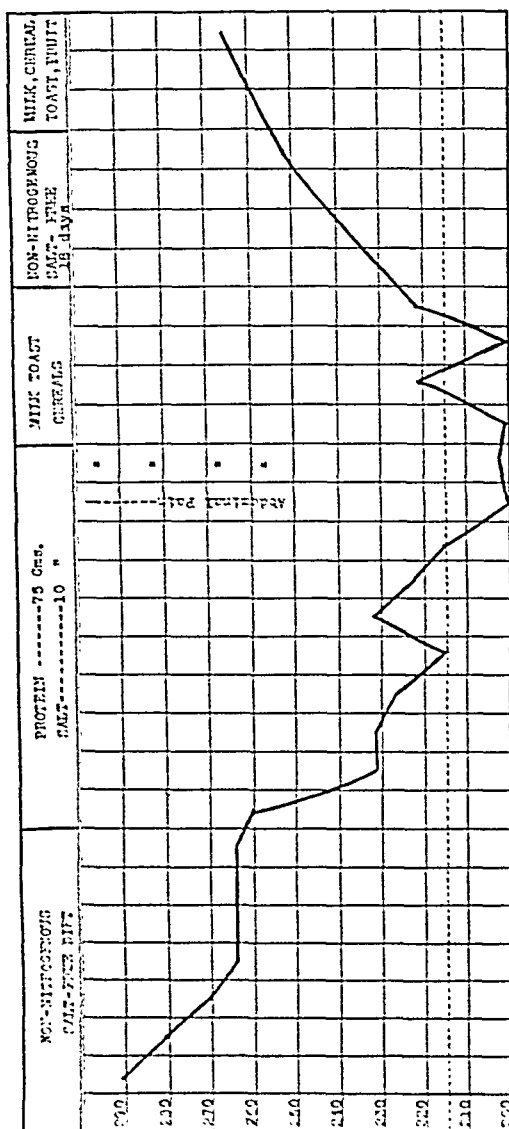
Fig. 56.—Systolic blood-pressure. Daily readings, showing relation to food intake.

nitrogenous one, the blood-pressure gradually could be seen to go up until on February 23d it reached a systolic of 270.

I am showing this case simply as an example of what one

dares to do in the way of dietetics under properly controlled institutional care. The fear of salt and the fear of protein in hypertension, it seems to me, have not been based on experimental facts. You will recall that recently Mosenthal[1] was unable to show any influence of protein in the dietary of patients with hypertension. My own clinical results when carefully controlled have likewise failed to show that restriction of protein or of salt intake has any definite effect either on the clinical course or on the blood-pressure of this group of patients. The rest in bed of the usual hospital régime probably does more than any dietary restrictions in improving the condition of these patients. I have seen at least one patient in whom every possible means was employed to reduce the blood-pressure, but the pressure constantly went up, until in despair the patient was left severely alone, when the blood-pressure immediately began to drop. It is not fair to say in this case that the fall in blood-pressure which followed the administration of a high protein and salt diet was caused by the food, although that conclusion is possible. It is absolutely fair, however, to say that in this particular instance the addition of large amounts of supposedly toxic food substance to the diet of a patient with high blood-pressure was followed by definite clinical improvement and by reduction in blood-pressure, and that subsequent removal of such foods was followed by a rise in blood-pressure.

The patient is shown mostly to call attention to the necessity of keeping a very open mind regarding certain dietary traditions which we are apt to follow. Recently I have had the opportunity of repeating the experiment on a similar case. I have seen definite clinical improvement following the administration of meat to certain subacute nephritis patients. In the latter case the generalized poor nutrition and anemia of the patient were probably responsible in part at least for a continuation of symptoms, and the improved nutrition following real food was actually responsible for the clinical improvement.

FATS IN THE TREATMENT OF DIABETES

Before showing you the next series of patients I think the time would be well spent in discussing the present status of certain phases of dietary therapeutics in diabetes. You may recall that it was not many years ago when Von Noorden and others were feeding large amounts of fats to their diabetics and when the dictum of treatment was to keep the patient well nourished. It is only within the last few years that, owing to the concentrated efforts of Allen, Joslin, and others, the dangers of fat to the diabetic were emphasized. Allen and Joslin in particular have advocated very strongly undernutrition as a fundamental matter in the treatment of at least most cases of diabetes. Joslin[2] recently has even gone so far as to say that "diabetes is largely a penalty of obesity."

The work of the American authors has been followed by tremendous good, and certainly by a diminution in the number of cases of diabetic coma, but it must nevertheless be admitted that the scheme of undernutrition as proposed by Allen is not by any means an unmixed good. In the first place it is doubtful if the extremely severe case of diabetes has an increased tolerance produced by undernutrition. That severely sick diabetics can be made temporarily sugar free much more rapidly and with much less danger following the lessons learned in the last few years is certainly true, but that there is final improvement in the condition of severe diabetics is doubtful. A great step forward was made in rationalizing diabetic therapy; but, as Allen[3] has stated, sparing the function by diet is a negative plan.

Of particular importance for practical feeding is the question of the proportion of fat to carbohydrate which can be given to a diabetic patient without danger of acidosis. Although this problem has received much attention, Ladd and Palmer[4] have again discussed this question. Their diabetic patients were given a diet sufficiently high in protein to maintain nitrogen equilibrium, and the proportions of fat to carbohydrate varied until the ketone bodies showed a definite increase in the urine. Using for available carbohydrate the total carbohydrate intake

plus 58 per cent. of protein intake, they find that the safe ratio
is approximately 1 to 4 in most instances. Newburgh and
Marsh late in 1920[5] showed that 73 cases of diabetes could stand
a fat-carbohydrate ratio of 5 or 6 to 1. This ratio does not
take into account the available carbohydrate or protein. They
used in their diet a low protein, giving only about 0.6 gram of
protein per kilogram of body weight, before the patient left the
clinic. They also showed that by increasing the protein during
the course of treatment, and leaving the fat and carbohydrates
alone, the patients would be glycosuric again. Their 73 cases
were not chosen, but were taken as they entered the University
of Michigan Clinic.

Before showing the cases which were studied following the
Newburgh and Marsh publication I wish to report one of an
old diabetic who entered the hospital on October 26, 1920,
and left on November 23, 1920. He was fifty-nine years old
and had been under my observation for the last ten or twelve
years. He belongs to a diabetic family, his mother and brother
having died of diabetes; one brother has gout; one sister has
uric acid kidney stones, and another brother a low-grade chronic
interstitial nephritis. During the course of our observation
the patient, who lived out of town, did not follow instructions
carefully, and his diabetes took a definite, downward course,
yielding rather easily to treatment when he was first admitted
to the hospital and becoming gradually more and more difficult
to render sugar free on subsequent admissions. For a week
before this last admission to the hospital he had not been feeling
well and had been confined to bed with pains in the leg, which
had been present for several months, frequent urination, in-
creased thirst, and, particularly for the last week, marked
drowsiness. When he entered the hospital he was in a semi-
comatose state and had a very definite tendency to drop off to
sleep.

He was thin and dry. His breath was heavy, but not sweet
of acetone. The right lung showed an involvement of about
a handbreadth in size in the lower axillary region, otherwise
the physical examination showed nothing of particular impor-

tance. His urine on admission showed approximately 4 per cent. of sugar and large amounts of acetone and diacetic acid. His blood-sugar was 0.40 per cent. The patient objected to any detailed studies being made on him, so that the blood chemistry and the alveolar air determinations were not possible.

The diagnosis of severe diabetes mellitus with acidosis bordering on coma was evident, and the patient was immediately put on a diet consisting of skimmed milk, water, coffee and tea, orange juice, and brandy. He was given enemas. Tincture of digitalis was likewise prescribed. No alkali was used. He began to show clinical and urinary signs of improvement in the acidosis almost immediately, and his diet was gradually changed. Owing to his rather considerable emaciation and the difficulty experienced at previous hospital admissions in rendering him sugar free, no real effort was made to make this patient aglycosuric.

His acidosis completely cleared up on November 5th, the tenth day after admission, at which time he was passing 1.8 per cent. sugar, or a total of 61 grams. Finally his sugar was reduced to ½ of 1 per cent., or 13 grams in a day. But the thing that surprised us most was that when he left the hospital his diet consisted of 100 grams of protein, between 150 and 160 grams of fat, and approximately 32 grams of carbohydrate. His blood-sugar was 0.20. His general condition was greatly improved and he was up and walking around with absolutely no clinical or urinary signs of acidosis.

We were struck by the fact that a patient entering the hospital in a severe diabetic acidosis could leave at the end of five weeks with a fat-carbohydrate ratio of 5 to 1 and not show any acidosis; or adding 58 per cent. protein, carbohydrate 90 to fat 155, with a ratio of 1 to 1.7.

CASE II

A lawyer, aged thirty-one, entered the hospital March 2d and left April 23, 1921. He had diabetes for eight years. His weight on admission to the hospital was 115½ pounds. In the course of these eight years he has had the typical symptoms

of diabetes and has been treated in many clinics throughout the country. Irritability, loss of strength, loss of vision, and edema of the ankles were pronounced. During the past three months he has been getting much thirstier than usual, and for the last six weeks he has been coughing frequently.

Physical examination was practically negative except for some pallor and considerable undernourishment. When admitted to the hospital he was passing 4.7 per cent. of sugar, a total of 84 grams. There was marked acetone and diacetic acid reaction in the urine. Blood-sugar was 0.38 per cent., carbon dioxid tension of alveolar air, 35.

This patient had been very carefully studied by other workers, and he was well versed in the whole question of the treatment of diabetes. An intelligent co-operation could easily be obtained. Undernutrition had not been successful, so we decided that he would be an excellent case on which to try a different method. However, instead of putting the patient immediately on a low protein and carbohydrate and a high fat diet, we placed him on eggs and vegetables for four days until he became very hungry, and without there being any marked change in his general condition or in his urinary findings. On the fifth day he was given a diet consisting of 17 grams of protein, 90 of fat, and 16 of carbohydrate, a total caloric value of 940 approximately. This diet was maintained for four days. At the end of that time the total sugar excretion was 7 grams. There was a trace of acetone, but no diacetic acid in urine. His blood-sugar had gone down to 0.22 per cent., and carbon dioxid combining power of the blood was 52 c.c. per 100. Carbon dioxid tension of alveolar air was 40.

The fats were then raised to approximately 135 to 140 and the carbohydrates to 27. This diet was continued for seven days, at the end of which time despite the fat-carbohydrate ratio of 139 to 21, which is over 6 to 1 (or including 58 per cent. of protein, 4.5 to 1), there was no acetone in the urine. Ammonia excretion was only 0.42 gm. and blood-sugar 0.20 per cent. At this time the patient was a little impatient because he was not sugar free, so his diet was reduced until he was receiving

only 15 grams of fat and 20 grams of carbohydrates. This was immediately followed by sugar-free and acetone-free urine. Then the fats were jumped to 90, the carbohydrates to 10, and the protein to 35. Additions were gradually made until, when he left the hospital on April 23d, he was receiving 17 grams of protein, 152 of fat, and 30 of carbohydrates. He had no sugar in his urine, no diacetic acid, and an occasional trace of acetone. On leaving the hospital he weighed 115 pounds, was without edema, and was feeling strong.

CASE III.—MILD DIABETES MELLITUS WITH ACUTE AND CHRONIC CHOLECYSTITIS

This man is forty-six years old and might be said to be a diabetic of the old school, as the first information obtained from him was that he had been a patient of von Noorden's and that fats were the best thing for him. He entered the hospital on April 13, 1921, on the service of Dr. Greensfelder, to whom I am indebted for the privilege of showing the case.

He complained on admission of severe pains in the right hypochondriac region, chills, fever, nausea, hunger and thirst, frequent urination, loss of weight, and boils on the back of his neck. He has had attacks of pain in the right hypochondriac region, with fever, chills, and nausea for the last ten years. He has known that he has had diabetes for fifteen years. The gall-bladder attacks came on about two or three times a year and were very severe and typical. His diabetic history likewise is typical and, as stated above, he has received much treatment. The boils on the back of the neck have been coming intermittently for the past few years. He has lost about 66 pounds in the last ten years. There is nothing else of importance in his history. When admitted to the surgical service of the hospital for his acute gall-bladder attack it was noted that there was considerable emaciation, exquisite tenderness, and a palpable mass in the gall-bladder region. There were a few boils on the back of his neck and some on his forehead. He had a temperature reaction varying between 99.8° and 102° F. and a leukocyte count of 17,000 with 77 per cent. polymorpho-

nuclears. The urine contained 7 per cent. sugar in the plain evening specimen voided on admission, with considerable acetone and a trace of albumin, the specific gravity being 1041.

This patient on admission to the hospital presented a problem of interest, a combination of diabetes and surgery, in which the surgical indication was for a condition independent of his diabetes. I shall not go into a discussion of surgery on a diabetic. The indications for treatment in this man were fairly clear. His diabetic condition was such as to make an operation undesirable unless urgent. It was decided to see what could be done by dietetic procedures accompanied by rest and catharsis and an ice-bag over the gall-bladder region.

He was put immediately on a diet of protein 50, fat 50, carbohydrate 50, since we always prefer not to make sudden changes in a diabetic's diet on admission to the hospital, and since before admission this patient had been indulging freely. This diet was kept up for four days, at the end of which time the patient was sugar free and acetone free. At that time the gall-bladder condition had pretty well subsided, the temperature had reached normal, and in the patient's own language "he was feeling like a new man." At first we thought that the gall-bladder complication would contraindicate a high fat diet, but in order to find out what would happen, on April 22d, which was ten days after admission and while he was still sugar free and acetone free, his diet was changed to protein 20, fat 136, and carbohydrate 25, giving a 5.5 to 1 fat-carbohydrate ratio (3.8 to 1 including available carbohydrate of the protein) with low protein and a total calorie value of 1400.

Protein was increased to 100, fat to 140, carbohydrates to 55 when he left the hospital—free of sugar and acetone and in excellent general condition.

CASE IV.—MILD DIABETES. OBESITY. HYPERTENSION.

I shall give you this case just in brief because she is a mild obese diabetic. She is a Russian Jewess, aged fifty-three, who entered the hospital on April 14th complaining of pain in the left thigh and inability to walk, epigastric discomfort, weakness,

and great thirst. She was in this hospital August, 1920 for an operation for varicose veins, and at this time sugar was not found in the urine. The weakness and excessive thirst had been present for about six months and was associated with polyuria, necessitating her getting up almost every half-hour at night.

I shall not go into great detail in discussing this case. The local condition in the left leg was apparently due to some disturbance around the acetabulum, the nature of which was not definitely ascertained.

The point I wish to emphasize in this case is that she is a stout woman, weighing 141 pounds, with a low-grade diabetes. On admission the urine showed 1.2 per cent. sugar, a total of 9 grams, no acetone or diacetic acid. The second day after admission she was given protein 20, fat 85, carbohydrate 14, calories 930. The blood-sugar was 0.18 per cent. The rest of the blood chemistry was normal. The blood-pressure was 190/85. Changes in diet were made first by increasing the fats to 135 to 140, carbohydrates 25 to 30. At but one time, the tenth day, a trace of sugar appeared in the urine, and occasionally a trace of acetone. On May 4th she was receiving 150 grams of fat, 40 carbohydrates, 100 protein. She still remains sugar free and acetone free.

This patient is a mild diabetic of the obese type in whom the high fat apparently has no harmful result.

CASE V.—SEVERE DIABETES MELLITUS, PULMONARY ABSCESS, AND HEMOPNEUMOTHORAX

The patient, a young woman aged twenty-nine, entered the hospital on April 8th complaining of cough, vomiting, weakness, and loss of weight. She says she has only been coughing a week, at first dry, later productive of a greenish, thick, offensive sputum. Vomiting started four days ago and has occurred a good many times, usually associated with severe coughing spells. For the past two months she has been becoming gradually weaker and is unable to walk up a flight of stairs without dyspnea. During the past year she has lost 81 pounds.

As far as she knows her diabetes is of one year's duration. There is no previous history of ill health. During the past year she says she has been on a diet.

On admission it was quite evident that we were dealing with a desperately sick woman. She was markedly dyspneic, with great emaciation, cyanosis, an annoying cough, and high temperature. There was a distinct acetone odor to her breath. Her breathing was of the dyspneic rather than the hyperpneic type, the respirations being rapid and shallow. The fingers show a low-grade pulmonary osteo-arthropathy. The lungs show marked dulness to percussion below the level of the fourth rib anteriorly and the midscapular region posteriorly on the right side. The breath sounds are absent. Tactile fremitus is absent and the voice sounds are distinctly nasal in quality. Grocco's paravertebral triangle of dulness was present on the left side. The note above the area of dulness was somewhat tympanitic. The heart was not enlarged or misplaced and the abdomen was negative. Temperature was 99.2° F., pulse 112, and respirations 32. Hemoglobin was 75 per cent., red cells 3,500,000; whites, 9000. Blood Wassermann was negative. The blood-sugar was 0.80 per cent.

Urine on admission showed a specific gravity of 1040, 3.75 per cent. sugar, total excretion 76 grams, acetone and diacetic acid were present, ammonia 0.56 per cent.

x-Ray examination of the chest showed a fluid level at about the third rib and what looked like a pneumothorax above that. Thoracentesis revealed a bloody fluid. Information obtained subsequent to admission revealed the fact that the patient's chest had been tapped about two weeks previously.

Owing to the acidosis the patient was put on skimmed milk, orange juice, water, tea and coffee, and as much bicarbonate of soda as could be gotten in. On the second day this amounted to approximately 100 grams. She began to pick up, and at the end of six days there was no acetone in the urine and her general condition was considerably improved. The temperature was still 100.2° F., pulse was quite irritable, varying between 80 and 100, respirations were steady at 24 to 28,

and she was considerably stronger. She was still passing 25 grams of sugar in the urine. Ammonia output was 0.28 gram. At this time her diet was changed to protein 50, fat 50, carbohydrate 50. Two days afterward she was given 20 protein, 85 fat, and 14 carbohydrate. On the third day after the institution of a high fat diet she became sugar and acetone free and except for an occasional trace of acetone has remained so up to date. Her diet has gradually been added to until she is now getting 100 grams of protein, 150 of fat, and 50 of carbohydrates, 1950 calories. Her blood-sugar on May 1st was 0.17. For the past four days she has been running a high fever, which reached in the evening 103.8° F., which apparently is associated with the pulmonary abscess. She is expectorating large amounts of foul-smelling pus. Despite this her urine remains free and her general condition is greatly improved. She looks entirely different from what she did on admission. Weight is increased. This patient certainly received benefit from the high fat diet, which would have been impossible had we adhered to the régime of undernutrition.

CASE VI.—DIABETES MELLITUS, CHRONIC NEPHRITIS, HYPERTENSION, AND CERVICAL RIBS

This patient is thirty-two years old. She entered the hospital on April 25th complaining of weakness, a gagging sensation in the throat, a numb feeling in the shoulders. Four months ago she noticed that she was unable to walk as much as she had been accustomed to on account of some slight indefinite gagging in her throat which was associated with a fluttering in the epigastrium and numbness in the shoulders. The numbness in the shoulders is particularly present in damp weather. She has known she had diabetes for several years and has had an increased appetite, thirst, and polyuria. She has been under treatment recently in two different hospitals. Three years ago she had erysipelas, during which time examinations of the urine showed the presence both of albumin and sugar. For the past three or four years she has been getting boils in various parts of the body.

Her mother was a diabetic and her father died suddenly of an unknown cause. Three brothers and sisters are living and well.

On examination she presents the appearance of a healthy woman, well built, and of a florid complexion. The supra-clavicular fossæ on both sides are filled by firm, bony structures, which were diagnosed as cervical ribs and confirmed by the Roentgen ray. General examination was negative. The heart was 12 cm. to the left. There was no edema. The blood-pressure was 145/95.

The urine on admission showed a good albumin reaction, few granular casts, 14 grams of sugar, and a trace of acetone.

Blood chemistry on admission was as follows: sugar 0.16 per cent., non-protein nitrogen 40, urea nitrogen 22.5, creatinin 1.3. Wassermann was negative. Alveolar air carbon dioxid tension was 45.

Owing to the combination of high blood-pressure, albumin-uria, and glycosuria this patient seemed to offer a particularly good opportunity for the study of a low-protein high-fat diet. She was immediately placed on a diet of 22 protein, 92 fat, and 20 carbohydrates, approximately 980 calories. There was an immediate disappearance of the sugar. The albumin gradually grew less, but did not disappear. The trace of ace-tone persisted.

However, after a few days the acetone entirely disappeared and the patient's diet reached 1800 calories. She left the hospital free from sugar and acetone. The albumin persisted and her blood-pressure on discharge was 140.

Discussion.—These patients are not shown to prove that a high fat diet should be employed in the treatment of all cases of diabetes. In the first place it must be remembered that the time of observation has been very short and the number of cases few. Diabetes is a chronic disease, and in order to prove the value of any method of procedure one must follow the cases not through a period of days or even weeks, but of months and years. It will be noted that in rendering a patient free from sugar we did not depart from the established procedure of diet

reduction. In the cases with acidosis fats were immediately removed. The results of this method of rendering patients sugar free seems so well established that personally I can see no advantage in adopting any other procedure in its place at the present time. Also, we did not continue all of our patients for a long period of time on a low protein diet, and it will be seen that the intake of protein could be raised considerably above the minimum of Newburgh and Marsh without producing glycosuria. Some of the cases were mild, and it is a well-known fact that mild cases of diabetes can be allowed considerable latitude in the choice of foods without any harmful results.

Our cases show that patients who come into the hospital in a severe state of acidosis may receive considerably more nourishment in the form of fat than we have dared to give in the past for years. It is seen also that, at least in my series, obesity, chronic nephritis, and hypertension did not contraindicate high caloric feeding. With both obesity and chronic nephritis I have found difficulty from long-continued undernutrition. The patient with pulmonary trouble who was so acutely ill on admission, but who passed the danger period under the usual methods of treatment, seems to me is definitely better off with the high caloric diet given than she would have been had we attempted a continuous period of undernutrition.

We must be very careful, as I have just said, regarding conclusions on such an important matter as the question of fats in the treatment of diabetes. Certainly at the present time it would seem undesirable to neglect the valuable lessons learned in the past few years. The results of Newburgh and Marsh and of the few cases I have cited, however, indicate that fat may be employed in some diabetics without the fear of danger. There are certainly some diabetic patients who do not improve greatly when undernourished. A higher caloric diet then might well be employed for such patients. This applies particularly to patients like my Case II, who had not reacted well to undernutrition after many periods of intensive study at different institutions, and who apparently handled

the higher caloric diet with ease and with considerable benefit to his general state of well-being.

The statistics on the diabetic death-rate, at least in this country, have shown a remarkable drop in the death-rate. Whether this is due entirely, as Joslin believes, to a more generalized use of undernutrition is perhaps open to doubt, for along with the general scheme of undernourishment came a period of more intensive study and more rational general procedures, not the least of which was the plan of securing the co-operation of the patient or the patient's family. It is perhaps important to remember that patients are much more likely to follow a diet which seems to appeal to them, such as a higher caloric diet, than they would one of undernutrition. The breaks which have occurred in the treatment of the individual case usually have occurred after the discharge of the patient from the institution, when he found it difficult to carry on for a long period with what he considered insufficient food.

In conclusion I should like to suggest that the treatment of diabetes is still not a simple performance with one stellar part. The rôles of protein, fat, and carbohydrate are still equally important.

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CLINIC OF DR. JESSE R. GERSTLEY

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL
FOR CHILDREN)

RISKING TONSILLECTOMY IN A THYMUS CASE

THIS little boy is three years of age. Well-developed and
rather tall for his age, he seems in perfect health. He is the
usual blonde type, with blue eyes and soft velvety pink and
white skin. The only striking abnormality upon inspection,
other than his open mouth, is the peculiar circle under each eye,
giving him an expression of fatigue. A marked bilateral cervical
adenopathy and similar marble-sized enlargements in the
axillæ and groins are revealed by the palpating finger. Percus-
sion of the chest reveals dulness extending two fingers to the
right of the sternum and up around into the second left inter-
space. Except for a very slight systolic murmur the heart
tones are clear. Blood examination demonstrates a slight
deficiency in red cells and hemoglobin, and a white count of
15,000, 65 per cent. of which are small mononuclears. Turning
now to his throat, we find it almost entirely closed by enormously
enlarged tonsils. Not only during sleep, but even at play and
at meals his respiration is seriously impeded.

The history of this child is unusual. Born weighing less
than 5 pounds, he was frightfully scrawny, weak, and emaciated.
Within a few hours attacks of apnea and cyanosis led on to
frequent convulsions which threatened to terminate his life.
A tentative diagnosis of atelectasis was established, for physical
examination at that time revealed nothing definite. The
heart tones were clear except for the murmur, which was still
present, but presently the x-ray (Fig. 57) showed a definite
enlargement in the mediastinum, extending up toward the
cervical region At this time there was a question whether

this was a congenital heart or a thymus, but subsequent history favored thymus. During the next six months these convulsions recurred at ever-increasing intervals, but always severe enough to leave him pulseless and in collapse. However, he gained normally in weight and seemed stronger in every way. In view of his apparent improvement no treatment of either x-ray or radium was inaugurated. An x-ray two months later (Fig. 58) revealed a shadow apparently increasing in size,

Fig. 57.—Roentgenogram taken at first examination showing a definite enlargement in the mediastinum, extending up toward the cervical region.
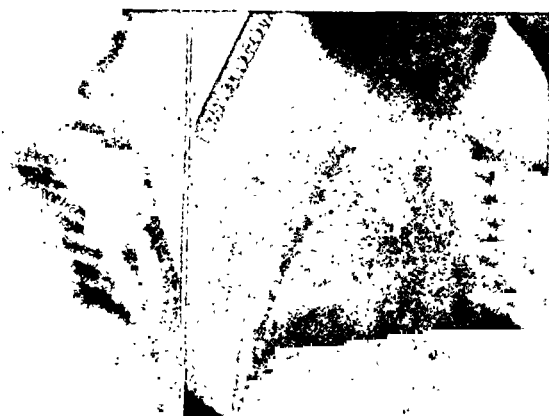
Fig. 58.—Roentgenogram taken two months later. Shadow is apparently increased in size, but the roentgenologist considered this due to a slight twisting of the child's body.

but the roentgenologist considered this due to a slight twisting of the child's body. No treatment was instituted, and from this time the child seemingly developed normally, showed no symptoms of any sort, and played actively and with the endurance and strength of a normal child.

Two years later the x-ray (Fig. 59) showed the shadow persisting, and there remained still the question as to whether this was a congenital heart or a thymus. Examination under

the fluoroscope, as previously, distinctly favored thymus diagnosis. There were no murmurs other than the very slight systolic impurity at the apex, which could be explained by the heart rubbing against the gland. There were never at any time signs of cardiac insufficiency. It was reasoned that if this had been a congenital heart, causing symptoms as severe as those of the first six months, the condition would not have improved so consistently. Lastly were the marked adenopathy and blood findings.
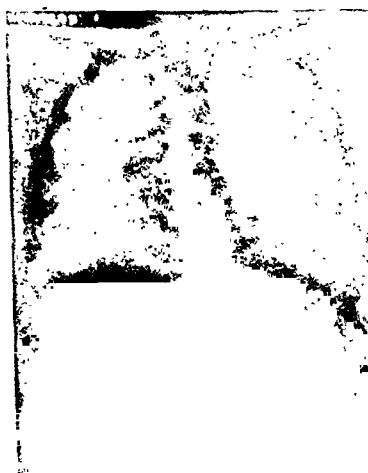
Fig. 59.—Roentgenogram taken two years later. Shadow in mediastinum still present.

Fig. 60.—Roentgenogram taken four months later. The shadow is considerably diminished, but still persists in the upper mediastinum.

Four months later upon the x-ray (Fig. 60) the shadow is considerably diminished, but still persists in the upper mediastinum. The child is now brought to us for advice as to tonsillectomy, for interference with respiration is so marked as to be a continuous source of difficulty. However, the parents know the danger of operation in thymus cases and are hesitant about taking the risk. What is to be done?

On the one hand, we have the typical symptoms of an en-

larged thymus and a great mass of data warning us that these children are subject to sudden death during operation. In many cases of unexpected death autopsy has revealed nothing but a hypertrophied thymus gland. How such an anomaly acts, however, is by no means certain. Some believe it due to an excessive secretion from hyperdevelopment and abundance of Hassall's corpuscles; others believe clotting blood in the heart and vessels prevents filling of the aorta; to others, there is a toxic myocarditis. Some have found chromaffin aplasia, some have not. Again, a thymic anaphylaxis has been blamed, and still others believe death due to a rupture of a hypoplastic cerebral blood-vessel. It is not my purpose this morning to enter upon a discussion of the functions of the thymus gland. It has been brought into almost every field of medicine, but even apparently scientific experiments are confusing. Some find an antagonistic action between thymus and thyroid. This is not unquestionably established, however. Some relate the thymus to phenomena of growth. Feeding the gland to tadpoles stimulates growth, says one observer. This is flatly denied by others. Extirpation of the thymus causes rickets, says one. "Rickets developed due to the unhygienic condition of the kennels," is the reply. It is supposed to have an internal secretion, but this has not been demonstrated. It may or may not be one of the blood-making organs. And so, when the whole matter is boiled down, we know very little of the thymus other than the tradition of sudden death. But let us go farther. Careful studies are beginning even to question this sacred dogma. For instance, Hammar in a series of cases found that many victims of sudden death had no enlarged thymus. Also the weight was subject to such variations as to make an estimation of the normal quite difficult. A review of the literature, too, from a somewhat critical standpoint makes one doubt that all cases of so-called thymic death are actually due to an enlarged thymus. For instance, it is not unusual for a physician called on such a case to sign the certificate as thymic death, but not to verify this by autopsy. From a scientific standpoint such conclusions are valueless, but undoubtedly statistics are based

upon many such diagnoses, *i. e.,* "A tradition tells us that children with large thymus glands are subject to sudden death; therefore, every case of sudden death must be due to an enlarged thymus." So our fear of this organ grows and grows. In view, however, of the uncertainty of anything absolutely definite concerning our knowledge, I believe that we are to some extent justified in shutting our eyes to the uncertain evidence behind us, and in considering a case like the present purely and simply upon its individual merits. On the one hand, we have a little boy who from birth has shown by symptoms and *x*-ray examinations a classical case of enlarged thymus. On the other hand, with absolutely no treatment this child has improved steadily, and if we did not know his past history nor his present *x*-ray and blood findings, we could undoubtedly call him normal. We are taught that this child if operated may die upon the table. Our medical experience tells us that with tonsils and adenoids such as he has, a scarlet fever, diphtheria, or virulent tonsillitis will probably carry him away. To which shall we subject him? One very excellent pediatrician in consultation has advised by no means to take the risk. Another one believes that we are justified. Personally, I believe in the view of the uncertainty of our knowledge of thymic cases, in view of the child's good health and strength, in view of his almost certain death if the throat conditions are not corrected, we should advise operation.

After-history.--The child was transferred to the Nose and Throat Department and subjected to the ordinary tonsil and adenoid operation. Blood examination made just before the operation showed the same lymphatic tendency. Coagulability was normal. He took a perfect anesthetic, bled very little, went through the operation just as would any normal child, and made an absolutely perfect recovery, with the exception that for twenty-four hours following the operation he was perhaps slightly more prostrated than the average. The operation caused a very great improvement in his general health, but had no effect upon his lymphatic blood findings or the adenopathy.

Six months later in an attack of pneumonia the temperature hovered for two days around 106° F., but he made a perfect recovery.

This case is reported to show that some children can take perfect anesthetics and survive operations in spite of enlarged thymus glands, and to emphasize that perhaps, after all, in forming medical judgments we must not be guided too strongly by hazy medical traditions, but must form our conclusions by careful, accurate history and study of each individual patient.

CLINIC OF DR. FRANK WRIGHT

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CAMOUFLAGED FOODS

IN the dietetic management of many diseases this fact is being emphasized by frequent clinical results, that it is far better to limit the amount of food, maintaining a relatively normal proportion of the various elements, than to unbalance the diet, thus introducing a non-physiologic food assortment into the regimen of a person already unable to properly assimilate certain food substances. A good example of this is furnished by the growing tendency to maintain a low caloric diet, approaching normal proportions with resulting weight loss, in many persons with diabetes, rather than furnish a diet of the requisite number of calories in which proteins or fat predominate. Likewise, a similar trend is evidenced in the food management of certain cases of arthritis and gastro-intestinal diseases. While highly desirable from the standpoint of the physician, this reduction frequently conflicts with the patient's habits of eating, his appetite, and its appeasement; interferes with bowel function, and when limited as to assortment frequently results in early abandonment, long before desired results are attained and enthusiastic co-operation of the patient enlivened.

To bridge over many of these difficulties we all resort to subterfuge, frequently in the open, with the connivance of the patient, as when we replace saccharose with saccharin in beverages and desserts; or in the case of bread-like concoctions which furnish bulk and transport butter, but either consist largely of non-digestible cellulose or are made of a porous protein framework perhaps of casein origin. Here the absence of

carbohydrate is stressed, but often we fail to go into detail as to what is present, likewise may we ask whether or not it is desirable. This is a reversal of conditions which formerly prevailed. You see we are becoming inquisitive and are no longer content to know the percentage of protein in gluten flour; we must know the carbohydrate content as well. Again, we substitute a hydrocarbon, using so-called mineral "oil," which has no nutritive value, in place of true vegetable oil in making our salad dressings, such as mayonnaise. Such instances furnish examples of palpable replacement, but in many others the diet changes are far-reaching, more subtle, and require greater care in planning and in interpreting the results which follow. It is concerning some of these practical problems I direct your attention this morning.

The use of saccharin as a sweetening agent is not to be advocated too freely. There is an accumulation of evidence which indicates that saccharin is not entirely devoid of deleterious action upon the human organism. Its use prompts a desire on the part of the one taking it to have sugar instead, and prevents the gradual diminution of the craving for sweets which one likes to see follow their continued curtailment. The taking of too large a quantity at one time often causes an aversion on account of the intense sweet taste or because of a metallic-like element in the taste effect which results in its disuse; as a rule, patients soon answer the question as to regulating its use by their own taste. It perhaps has its best sphere in the sweetening of certain egg-and-milk combinations, frozen puddings, and stewed fruits.

In the selection of bread substitutes there is reason for making a choice quite apart from the comparative freedom from starch which they all have in common. When we take into consideration the frequency with which infections, or histories of past infection of the upper digestive tract in patients with diabetes, where duodenitis, subacute pancreatitis, and catarrhal cholecystitis have been or still are present, these frequently associated with putrefactive conditions in the small intestine and colitis or constipation, one hesitates before using a bread

substitute of protein composition, such as gluten or casein, which increases the amount of putrefaction and may augment the pathology.

It is here that we are allowed a choice in the selection of our camouflaged bread substitute based upon a fact which, to date, has not received the recognition I think it should. Some time ago our Dean, Dr. Arthur I. Kendall, published the discovery that many pathogenic organisms elaborated their toxins only in the presence of protein; that if carbohydrates in the form of the various sugars predominated these, some organisms failed to produce these toxins, thus losing their pathogenic properties. As he put it, in the presence of the proper sugar these virulent organisms become ordinary milk-souring bugs. Applying this fact to the bacteriology of the intestinal tract we note that the sugars disappear quickly from the upper portion of the canal, being absorbed promptly, so that their action cannot be counted upon as persisting for any great distance along the canal. On the other hand, we are quite familiar with the use of coarse brans and cellulose-containing vegetables in the treatment of constipation, likewise the gas production which they elicit and the type of stool which frequently follows.

That these bran and cellulose products pass through the whole length of the tract is likewise well known, but that they lose at least some portion by disintegration brought about by digestion, or more probably by bacterial action, is less frequently recognized. We are less interested in the fate of the coarse cellular carbohydrate with its imprisoned starch granules than in its effect upon the bacteria which it meets while traversing the intestinal tract. That the action is often beneficial in combating the organisms I firmly believe, and for that reason should guide us in choosing cellulose products, which, after all, are only refined sawdust, well-washed bran, or bran and soy bean muffins in the treatment of many of our cases of diabetes.

Continuing the subject of foods for the diabetic, it is when we consider the use of vegetables that we find that as physicians

we have deceived ourselves and not the patient; this so frequently that we may well take time to become somewhat better acquainted with them.

The classification of vegetables into groups of 5- ,10-, 15-, and 20-per cent. carbohydrate content emphasizes the fact that they do contain sugar-producing substance in varying amounts, and we choose the ones containing the lesser amounts because of the *lack* of the one constituent; potentially they are less liable to cause trouble, so to speak. But do they have any positive value? Are we justified in stressing the utility of these articles of diet in an emphatic way which the patient will grasp, and so enter upon the use of these vegetables with some degree of enthusiasm? These questions may be answered in the affirmative. Vegetables are required for their vitamins, for their bases, which form salts when combined with the acids formed in the course of metabolic activity, and for the calcium, iron, and potassium which they furnish.

When a person is on a mixed diet the eggs and meat are distinctly acid-producing foods; in diabetes, particularly, the fats are frequently incompletely oxidized, with the resulting formation of quantities of ketone bodies, acetone, diacetic acid, and beta-oxybutyric acid. If, then, the diet of a person with hyperglycemia contains a predominance of these foods, it follows that a commensurate amount of base should be included to neutralize the acids formed in the course of metabolism, the base being furnished by vegetables rather than by administering alkalies in the form of sodium bicarbonate and chalk, a practice which formerly had more advocates than at present.

A careful study of the composition of vegetables of low carbohydrate content will demonstrate that these same foods contain a relatively large amount of base or potential alkali and, what is more, that the assortment is much more suitable for the needs of the body than the alkali mixtures usually prescribed. The interesting finding that a hundred caloric portion of potato contains an amount of basic substance sufficient to neutralize a hundred calorie portion of beefsteak will prompt us to furnish a part of the daily allotment of carbohydrate in

the form of potato, which provides base and salts not supplied when wheat is eaten. The well-recognized deficiency in calcium of practically all diets is another reason for utilizing vegetables in abundance, for these same vegetables are rich not only in calcium, but iron, potassium, phosphorus, and sulphur as well. Lastly, the presence of vitamins is a sufficient reason for prescribing green vegetables freely, even though no other needs were met by their ingestion. If these interesting and highly potential substances are required for normal growth and nutrition, how much more are they required in pathologic states of metabolism.

Substitution of nuts and legume proteins for meat in the form of meat-like loaves is practised extensively. As the term "meat substitute" is often used, it is well to note that Sherman says that the nuts existed first, and it is more logical to speak of meats as "nut substitutes" instead. The fact that the proteins of soy bean and Brazil nut rank with those of milk and egg as being complete proteins capable of maintaining adult nutrition and providing for the growth of the young prompts one to view with friendly interest the vegetarian who advocates "mock meat." Other meat products which can be counterfeited are the soups made from potato-water and others made by long boiling to disintegrate yeast. The latter, when properly prepared, is practically identical in flavor.

To attempt to follow the various fat modifications which are used in place of butter, lard, and olive oil it is well to state that, as a rule, the vegetable products lack the necessary quantity of vitamins. The vegetable butter substitutes are particularly to be avoided in the feeding of children who require all the constituents of the butter-fat. Other than that the main interest is concentrated on the fact that many of our American produced vegetable oils are in every way quite as desirable as olive oil of unknown origin. It is well to remember that Italy is one of the largest buyers of our vegetable oils, and at times has exported more olive oil than it appears she could produce. Blended oils were more frequently found a few years ago when the methods of detection were first stringently applied.

We even extend our therapeutic camouflage to the use of acids and bases by using citric acid in place of vinegar, where the primary acid taste is followed by an alkaline reaction when the acid is oxidized; to the use of phosphoric acid in acid phosphate mixtures upon salads when we wish to modify the reaction of urine. To combat the lime deficit so frequently found the salt-cellar is provided with a mixture of salt and calcium carbonate.

THE USE OF THE ENDOCRINES IN THE TREATMENT OF CERTAIN GYNECOLOGIC DISORDERS

I DESIRE this morning to present a few cases demonstrating the use of the endocrines in the treatment of certain gynecologic disorders. Inasmuch as endocrine dysfunction is represented by physiologic disturbance, at least in the larger measure, it follows that the therapeutic use of extracts made from ductless glands must be applied chiefly to disorders that are physiologic in nature rather than pathologic. It is necessary, therefore, in order to obtain the best results to carefully select cases that are to be treated on this basis, that is to say, patients suffering from pathologic lesions of the pelvis, such as inflammatory reactions, tumors, etc., would not be benefited by endocrine therapy, but must be treated, as in the past, by surgery. I make this point in order to emphasize the importance of confining endocrine therapy to its proper field, which for the present is a limited one.

Probably the commonest expression of endocrine disturbance lies in menstrual irregularities. Only too frequently do we find, particularly in young women, patients suffering from amenorrhea, dysmenorrhea, and, more important yet, excessive bleeding associated with menstruation.

Some of these individuals, perhaps we might say the majority of the young girls who have these menstrual irregularities during adolescence, straighten up and become normal when they reach the age of nubility, eighteen, nineteen, twenty, sometimes not until they are twenty-three, and occasionally not until they are twenty-five. There are, however, a certain proportion

who continue to be irregular for years in the form either of an amenorrhea or a condition of excessive bleeding, and it is this group that chiefly comes up for treatment. Today we see not a few young women who are bleeding excessively and who require treatment even before they have reached complete development.

Another expression of dysfunction during the years of adolescence is dysmenorrhea, pain associated with menstruation. Dysmenorrhea is, again, a thing that in young girls is too often neglected by the parents. They assume that this also will right itself when she becomes older and the periods are better established, or, as is too often the case, marriage is awaited with the idea that in some mysterious way this is going to relieve dysmenorrhea. A generation or two ago there was not such a long wait, because it was the fashion for young women to marry early. That is not true now, in many instances at least, so that the hope of marriage as a cure of dysmenorrhea is a thing not so anticipated as it used to be.

Now this dysmenorrhea is of the uterine colic type, that is, the menstruation is characterized by severe cramping pains, sometimes so severe as to incapacitate the individual for a day or two or even three, during which time she has to go to bed on account of the pain. Therefore it becomes a definite pathologic entity, because any affection, call it illness or what you will, that prevents a person from living a normal life—and a normal life is not going to bed on account of pain—prevents her from doing her work, and it makes just as much difference to the girl who is going to school as it does to the girl who is working for her living in the shop, factory, or office. In adolescent years it is too often neglected. In postadolescent years it is also too often neglected. Dysmenorrhea is so common that it is taken for granted, and the majority of women, especially the unmarried, regard it as something they have to endure, something that is normal. They regard those women who pass through their periods without pain as unusually fortunate. Of all the disturbances of menstruation in early life, dysmenorrhea is far and away the most common, and it is the one that is the most neglected. Women do not come for treatment

because they do not like to have to consult a doctor. It is usually pain in the last analysis that brings the individual to the doctor, or discomfort of some sort or another, and it is not a pleasant prospect for a young woman to have to consult a doctor for the first time in her life, particularly with reference to pelvic disturbances. Her parents do not bring her or she refuses to go, putting it off from month to month or from year to year, with the idea that it will be nothing. Dysmenorrhea, however, is a disturbance that does not tend spontaneously to right itself. It is a condition that tends generally to be prolonged beyond the years of nubility and carried into the more advanced years of adult life. Farther on, after twenty years of age, for instance, we find again evidences of a purely physiologic disturbance, and from what I have told you about menstruation you have gained the idea that every period of ovarian activity is in a mild way somewhat aggravated, or is at least an expression of endocrine disturbance.

Amenorrhea is not so common; it is not often seen, at least, in the years of sexual activity, and particularly is that true of the individual who is married and has children. There is something about pregnancy no doubt that helps to stimulate menstrual function and to improve it in many ways. Whether it is the fact that the ovaries have periods of rest for ten months at a time, or whether it is the influence exerted upon the general metabolism by the hormones that are produced in pregnancy, for instance, by the trophoblast, which acts in a sense as a ductless gland, or whether it is the function of the breasts in lactation that assists in rendering the individual more normal, we do not know. There is reason to think that pregnancy physiologically is a good thing for an individual, so that after pregnancy we see an improvement in the character of menstruation, and menstruation is the first outward evidence of endocrine stability. We see women who have been irregular become regular after pregnancy, and those who have bled excessively tend to bleed more normally, those who have periods of amenorrhea tend to bleed more regularly; yet there are those individuals who again develop amenorrhea in the ab-

sence of pregnancy, periods that become less in amount and duration. That is particularly apt to be expressed in the unmarried women who work, especially those who work at some routine vocation, such as school teaching or office work, particularly if they are not very robust at the beginning, the undernourished type of individual. These individuals show a deficiency in constitutional development from the beginning. They show early fatigue. One of the expressions of this is a decrease in menstrual capacity—a relative amenorrhea. It is seen also in the excessively obese. Here there is, of course, a suggestion of myxedema, though it never arrives, yet maybe it does arrive later in midlife, at which time myxedema is most commonly seen.

After adult age, especially if the individual has had children, menorrhagia or metrorrhagia, excessive uterine hemorrhage, is much more apt to be due to local faults, such as the development of tumors or of misplacements. Inflammations, tumors, and displacements are the three chief factors in excessive uterine bleeding after the individual has reached adult age, and especially if she has had children.

In treating cases of this sort the first idea is that if the patient does not menstruate sufficiently well, the trouble is in the ovary. It is an ovarian dysfunction, a hypo-ovarianism, and the thing to do is to give the individual ovarian extract of some sort or other. Years ago, before our present knowledge of the endocrines, these individuals were treated in just such a way, but with disappointing results. Men had shown that they could treat myxedema successfully by giving the patient thyroid extract, and so they thought that they could treat amenorrhea by giving the patient ovarian extract, and they were disappointed; hence this treatment fell into disuse, or it never came into successful use, and was not popularized.

Likewise, too, excessive hemorrhage, excessive bleeding may be regarded as due to excessive activity of the ovary, and therefore, if you give something to gain a balance, you benefit the case—some other ductless gland for instance.

First of all, before we go any farther into the therapy we

must, of course, select our cases with a great deal of care, and we must rule out the cases in which we know from years of experience that pathologic conditions might produce the symptoms that were complained of. That is to say, first of all we must examine the patient. We must be sure that she has no tumor—either uterine or ovarian. We must be sure that she has no displacement of the uterus. Displacement of the uterus is not so rare in the nullipara. We must also be sure that she has no general systemic disease that is a factor in amenorrhea or excessive bleeding. That is to say, she must not have tuberculosis, anemia, hookworm, or any of the other wasting diseases. She must not have heart disease, because heart disease is a definite factor in excessive bleeding. At no time in your life must you consider a case of excessive uterine bleeding without a careful study of the heart. Stenotic lesions in particular are apt to produce uterine hemorrhages. Likewise, diabetes, cirrhosis of the liver, and conditions of that sort must be ruled out. Fortunately, in young women these constitutional lesions, except heart disease, are not so apt to be found. Of course, we do find diabetes in the young and we do see menstrual disturbances associated with it. After we have had years of experience in this line of work and we come to analyze our cases we find, in the last analysis, that but few cases may be regarded as purely physiologic disturbances; just as when you come to study the menopause you find so many things that could be associated with the climacteric by the time the individual is forty-eight or fifty years of age, that when you have ruled out all pathologic lesions you have very few purely physiologic climacterics left.

The conditions that are most frequent in adolescent years and that require treatment are the menorrhagias and dysmenorrhea. Now I want to cite 2 cases of dysmenorrhea to show you how it works out. The first one is a young woman, eighteen years of age, a student at boarding-school and raised in a type of our best American families. She began menstruating at twelve years. She menstruated every twenty-eight days for five years. Her complaint was pain, with every period so

severe that she was compelled to go to bed, associated with some nausea. When she was fifteen years old this was so severe that her parents took her to a surgeon, who did a dilatation and curetage, with the idea of relieving the pain. Dilatation and curetage is one of the old treatments that has been in practice for years for the treatment of dysmenorrhea of this type, and it is based upon the idea that there is an obstruction in the cervical canal, or a kinking, that produces a physiologic obstruction if not an anatomic one, and as a result of which the blood clots before it escapes from the uterus and the expulsion of the clots causes pain. I think you all understand that theory. The dilatation is really somewhat more than a dilatation, it is a divulsion. The cervix is dilated with Hegar dilators until you can introduce your finger into the cervix. It is practically impossible to dilate a non-pregnant cervix more than that without breaking its walls in some degree. Every break means scar tissue healing. This operation is done for dysmenorrhea and sterility, and it is fairly successful as a method of curing sterility in a certain proportion of these cases. The patient is generally in the hospital a week, goes home, and she does not menstruate the next month because she has become pregnant. Then she has no more dysmenorrhea because she is pregnant for ten months, after which labor produces a physiologic dilatation of the cervix that certainly cures dysmenorrhea.

No woman who has had a child should have dysmenorrhea. If she does, there is something the matter locally, either a tumor, inflammation, displacement, or something of that sort. If pregnancy does not take place in the married woman after dilatation, then, of course, the dysmenorrhea is not relieved permanently.

One of the reasons why dilatation and cureting fell into disuse as a treatment for dysmenorrhea in these young girls is that it relieved them only for a few months. They would have no pain for one or two months, and then it would return just as severely as before.

If this young woman had not given a history of having had this operation I might have been tempted myself to suggest

it. I examined her and found nothing whatever pathologic in the pelvis. The uterus was upright, it was anteflexed rather sharply, but that is normal in a nullipara. The appendages were negative—perfectly free and in position. There was no evidence of inflammation and no evidence of tumors. Therefore I wrote a prescription for corpus luteum. Corpus luteum is the dried extract of the corpora lutea of pregnant animals obtained from the stock-yards. It is taken either from the sheep or the cow or from swine. This patient was told to take 5 grains of corpus luteum three times a day for five days preceding the next period and every period thereafter.

I had a letter from this patient in three months. At the next period, she wrote, she had to go to bed for one day, but she did not feel very badly and was not nauseated. The second period after that she did not go to bed at all. She had no cramps and no nausea. At the third period she awoke in the morning and found that menstruation had taken place during the night. I saw her again this spring at her Easter vacation, and she had had no dysmenorrhea since, but she has kept on taking the medicine.

Now I do not know why corpus luteum relieves this type of dysmenorrhea. We do not know enough about endocrine interrelation to explain a thing like that. It is pure empiricism, and empiricism is always ahead of knowledge. I certainly have never regarded that type of dysmenorrhea as due to hypo-ovarianism. This young woman was well developed, was the picture of health, and offered no reason for thinking that the ovaries were deficient.

My second case is that of a young woman, twenty years of age, working in an office as a clerk. She was brought to me by her aunt, upon whom I had operated for an ectopic pregnancy. She began menstruating when she was fifteen, periods coming every twenty-eight days for five or six days, with severe pain, so that she also had to go to bed for three days every month. She had to stay away from work for this length of time every month. Dysmenorrhea is a definite factor in industrial medicine, particularly evident in large institutions where

many women are employed. Statisticians who have studied this phase of the subject have told us how many days are lost to labor in a single institution where many women are employed by dysmenorrhea alone.

She also had nausea and in addition to the dysmenorrhea she had a constant leukorrhea. When I examined her the pelvis was normal except for uterine retroflexion. The cervix was up behind the symphysis, the corpus in the culdesac of Douglas, and the appendages prolapsed. The organs were so free that I had no difficulty in elevating the uterus and putting in a small pessary, such as is designed for the nullipara. I did this in order to ascertain what influence the displacement had on the dysmenorrhea. She came back after the next period and said she had just as much pain as before and had to stay away from work for three days, but the leukorrhea had stopped four days after the insertion of the pessary, an interesting observation. I then wrote her a prescription for benzol benzoate. She came back after the next period and said that it had come at the regular time, there had still been no leukorrhea, and that she had started in menstruating with the same severe pain that she had had before. She then took the benzol benzoate soluble elastic capsules, had some relief, but she still had pain for three days and was away from work for three days.

I then told her to take the benzol benzoate two days before she expected to be sick. She did this, and thought the pain was considerably less, but still she had pain for three days. I then told her to stop the benzol benzoate, and I wrote her a prescription for corpus luteum. The next time she came back she reported having had pain on one day for three hours, not very severe, and there were no clots. She kept on taking the corpus luteum, 15 grains a day for five days prior to each period. It is always easy to give it where the periods are regular. She has had no pain since. I saw her two days ago (May 5th). She now menstruates without any pain at all and does not lose any time from work as many women do who have much dysmenorrhea.

I did not want to keep the pessary in too long, so I removed

it in March. I removed the pessary first to see if the uterus would stay in place; and second, to see if she would have a return of the dysmenorrhea or the leukorrhea. She came back the next month with the uterus again upside down and the leukorrhea again present. The leukorrhea came on a week after the pessary was taken out, but she had no dysmenorrhea that month, proving that the displacement of the uterus was not a factor in the dysmenorrhea and that it was the cause of the leukorrhea. I then put the uterus up in place, reintroduced the pessary, and the leukorrhea stopped as before.

There are theories to explain the rationale of this treatment, but none answers every phase of the question. The corpus luteum used in these 2 cases was that prepared from the corpora lutea of sheep and cows. I have used that prepared from the corpus lutea of pregnant swine, and it seems to work quite as well.

Next I wish to tell you about 2 cases of amenorrhea in young unmarried women. The first patient is twenty-two years of age and a student at the Art Institute. She is a slender young woman somewhat under weight, with the physique of a girl of seventeen or eighteen. She started menstruating at sixteen, a little late. She menstruated regularly every four weeks for five days until eighteen years of age, when she stopped, and has not menstruated since. In the summer of 1920 she was treated for this with corpus luteum, and during that time she had two indefinite periods, a white discharge that lasted two or three days. When she came under my observation she was under the care of an internist in the Presbyterian Hospital, suffering from food eructations, not nausea, but merely eructations of undigested food. This patient had a retroverted, retroflexed uterus, the pelvis being otherwise normal. The only thing in her history that I could see that would have a bearing on her case was the fact that her mother suffers from migraine. We do know that migrainous women tend to have children who have migraine or sometimes more marked disturbance of a nervous or neurotic type. I merely mention that as a point that might be of interest. Some men believe

that migraine has an endocrine disturbance as a basis, while some scoff at that idea. This patient previously had been taken by her parents to one of our large clinics, where she was told that nothing in the way of a surgical operation would avail, because putting the uterus in position would probably not influence the amenorrhea. All of these things added to the interest in her case. Amenorrhea is much more important than dysmenorrhea, because it suggests the loss of function of organs that are peculiarly important to young women. The idea of premature menopause is not at all pleasant. The patient did not show any evidence of the climacterium; she had no vasomotor disturbances; she was not particularly nervous and unusually free from "temperament."

Here again I elevated the uterus and put in a pessary. The uterus was perfectly free and normal, and the appendages were negative. I gave her extract of the whole pituitary gland, 2 grains three times a day.

She came back three weeks later and said she had the white discharge for five days January 7th. Then a week later (January 12th) she bled for five days—red blood. On February 5th, which was only three weeks, she bled for six days—red blood, without pain. On March 4th, one month later, she bled for five days without pain. On March 20th she bled again for four days without pain. She was menstruating, in other words, every three weeks, which was a little better than anticipated. I told this patient she would have to take this medicine for three months at least, possibly for five or six. I did not give her any hope. I did not talk very much about making her menstruate. I then cut down the medicine to two doses a day and told her to stop taking it when she was bleeding. She bled again the middle of April. She was menstruating again when I saw her May 5th.

This is one of the best results I have ever seen. These cases do not all work out so well. The employment of pituitary gland is based on the idea that the ovary is not the strongest link in the endocrine chain, but is an incidental factor. It comes in at puberty and it goes out at the menopause, and it is

influenced in function by the stronger elements in the endocrine system. It is stimulated or inhibited by the more important links in the endocrine chain, chiefly the pituitary, the adrenals, and the thyroid. In a sense, therefore, this case was treated on the basis of a hypopituitarism.

Another case is that of a young woman of twenty-three which shows both amenorrhea and menorrhagia. The patient began menstruating at twelve, every five weeks for four days without pain, until she was eighteen. For the past eight years she has been irregular, menstruating only two or three times a year—March, October, and November, 1920, for two days, and twice in 1919 for two or three days. If this girl were extremely obese, if she were lethargic, if she were happy-go-lucky in her disposition and did not want to go to school, we would say "hypopituitarism—Fröhlich's syndrome," but she is not a Fröhlich's syndrome. On the contrary, she is slender, a bit undernourished if anything, extremely bright mentally, and very capable. She also works in the Art Institute, not only as a student, but teaches. I give all this data to show she is not a Fröhlich's syndrome. I have never seen a Fröhlich's syndrome that was paying her way through school. This young woman I also put upon whole pituitary gland, 2 grains three times a day. This patient menstruated on December 3d for two days, red blood, on January 10th for two days, and the 14th of February and the 14th of March. I saw her this month, and she had had another scanty period in April without pain. The periods are scanty, but she is menstruating under this treatment.

As to menorrhagia, I have 2 cases for illustration, in women of twenty-one and nineteen respectively. These are peculiarly good cases and excellent problems for study. The young woman of twenty-one began menstruating at fifteen, every five weeks for five days without pain. For three years, since she was eighteen, she has menstruated every two weeks for from seven to fourteen days. There were some months when she was bleeding every day; there was no pain and no clots were present. A dilatation and curetage had been done in 1919. Dilatation

and curetage is the cure for all menstrual disturbance according to the old treatment. When she first came under my observation I gave her a preparation that is well advertised in the medical journals, a preparation of endocrine glands that is advertised as a cure for everything from dysmenorrhea to the menopause. It is a type of the old-fashioned shotgun prescription. We must use these preparations in order to ascertain their value. She took it three times a day for a month, with no perceptible improvement. Then the next month I gave her 2 grains of the whole pituitary body three times a day. After that she stopped bleeding for three weeks, then bled for four or five days freely, and stopped. The tendency to bleed profusely at the intermenstrual period entirely stopped. Incidentally, she also had a displacement of the uterus; aside from that there was no other evidence of fault in her pelvic organs. Now, of course, displacement of the uterus is set down as a factor in excessive bleeding, so before I started her on the treatment I corrected the displacement with a pessary. Since then I have operated upon her, performing a shortening of the uterine ligaments. But before I operated I succeeded in stopping the intermenstrual bleeding.

The 2 cases of amenorrhea were corrected by giving pituitary body. Now here is a case of excessive bleeding that was benefited by giving the same medicine in exactly the same way. This would appear to be inconsistent on the face of it, but I have already explained that the ovary works accordingly as it is stimulated or inhibited by the more important glands in the endocrine system. In the case of amenorrhea the ovary was inhibited; why would you give pituitary gland to inhibit it further? In this case the ovary was overactive; why would you give pituitary gland to overstimulate it further? That is not the argument. The argument is that in most of these cases of menstrual disturbances the ovary is not primarily at fault, and when you give pituitary gland it is apparently a regulator of the whole system. It is like the governor on an engine; if you take it away the engine will run wild. That is the theory upon which treatment is based. It does not always

work, which is all the more reason for doing what I said in the beginning—in selecting cases for treatment we must very carefully rule out all other factors which might be the causes of the disturbance.

Another young woman of nineteen years began menstruating at fifteen, every four weeks for seven days without pain. For the last four years she has had excessive bleeding without clots or pain. In 1920 she missed three periods—September, October, and November. She also is a student from the southwest. After coming to Chicago last year she missed three periods. One of the conditions recognized as a factor in amenorrhea is a change in the environment. Two years ago this patient also had a curetage for excessive bleeding. That is to say, this excessive bleeding began two and one-half years ago, and after it lasted for six months she submitted to dilatation and curetage, with no relief. When I saw her she had been bleeding since February 28th. Two days after she started taking pituitary gland she stopped bleeding. She menstruated in three weeks from that time, a little too soon, and a profuse period which lasted six days. When I saw her again yesterday there had been no further menstruation.

The type of case described by the phrase "Fröhlich's syndrome" represents a most unsatisfactory condition to treat. This patient menstruates but two or three times a year, and then scantily. She is extremely obese, of a happy-go-lucky disposition, does not take to education, does not like to go to school, can never keep up intellectually with girls of her age, and when married makes a perfectly happy wife. She makes a good cook, loves to eat, and is so perfectly satisfied with life that she is twenty-five or twenty-six years of age before she comes for treatment, the complaint usually being sterility. She represents hypopituitarism of a definite type, and this is the patient to whom ovarian extract used to be given without benefit. It is not the thing to give them. I have had a number of these patients, and years ago, before I had found out what the fault was, I also treated them with ovarian extract without influencing them in any way. We recognize it now as a hypo-

pituitarism, but treatment is, as I have said, very unsatisfactory. If we could get this individual while she was a girl, if while she was fifteen or sixteen years of age, we could look forward ten years and see what she was going to be like; if then we gave her hypophysis, we might do her some good.

Then we have another type of woman that I have already hinted at—the woman who works and who tends to take on weight, but never becomes markedly obese. She is easily fatigued and has low blood-pressure—the flabby, soft type of individual. She is normal intellectually and capable in every way, but she has to drive herself in order to accomplish what she has been accomplishing in the past. This is the type of woman that breaks down and goes to the sanitarium, often under the diagnosis of "nervous prostration." If they happen to be married and have well-to-do husbands the sanitariums are fortunate, because they always get them. These patients show a relative amenorrhea; they menstruate scantily and come into the menopause early. They are medical cases, not gynecologic; that is, the internist or neurologist sees them oftener than the gynecologist, because they are often normal pelvically. I have also treated this condition with hypophysis. I give them the whole gland in preference to either the anterior or the posterior lobe after they have reached adult age, watching the blood-pressure carefully during the treatment. In both this case and the one previously referred to thyroid extract is also well indicated, either alone or with pituitary extract.

This brings us up to the climacterium. While this condition is extremely protean in its manifestations, exhibiting a great variety of symptoms, varying greatly in degree in different cases, we find there are practically only two types to treat. We do not have to treat the woman who is going through the climacterium without appreciable reaction. If she is not showing vasomotor disturbances of depression, I believe we should let well enough alone and let her go on and lead as normal a life as she can. There is one type, with vasomotor symptoms, whose reaction suggests hyperthyroidism. She is the one that responds to corpus luteum. Some clinicians give these patients

what is called "ovarian residue," which is extract of the whole ovary, with or without the corpus luteum. This is done upon the theory that not only is there a hormone derived from the corpus luteum, but also one produced in the so-called interstitial cells of the organ, and that a better result is therefore secured by feeding your patients extract of the whole ovary, or ovarian residue, than you do by treating them with extract of the corpus luteum alone. Other clinicians have found extract of the corpus luteum of pregnancy entirely sufficient.

These patients are told when you start treating them that they must take the medicine right along for a continued period of time. However, I explain to them the difference between the climacterium and the menopause, and give them some idea as to what the climacterium is and what it means. Then I tell them that, as a rule, according to my observations, if the climacterium is treated, it does not last as long nor is the reaction so pronounced as if it goes untreated. It will run along through a period of three to five years if it comes on at the normal age of forty-six, forty-seven, or forty-eight, and sometimes longer than that. I also explain to them that we may not be able to stop the vasomotor disturbances altogether, but that we can markedly alleviate them. Then, after having had the treatment for three months, I tell them to stop it and see what happens. So far as my experience goes every patient who has been treated on this basis has returned and asked that the treatment be renewed. Thus far I have not found it necessary to treat a patient longer than eighteen or twenty months.

The other type in the climacterium is the patient who shows the picture that points toward a myxedema, that is, a hypothyroidism, the patient who is hypotonic, who has a low blood-pressure, sometimes markedly low. They show a systolic pressure of 115, 110, or 100, and in one case as low as 98. The diastolic pressure is not lowered in proportion, with the result that an extremely low pulse pressure is present—15, 20, or 25. You cannot expect such a patient to have very much energy. This is the type of woman who is thoroughly exhausted; she cannot do anything; it makes her tired to get the children ready

for school in the morning. She cannot get the meals and is fatigued all the time. She may be well nourished. I am not talking about enteroptosis, even though the symptoms are somewhat similar. This patient, like the case of Fröhlich's syndrome, is a most unsatisfactory one to treat. Certainly you would not give this patient corpus luteum. We treat her by giving extracts of the pituitary and thyroid.

Before we leave the climacterium I want to say a word about premature menopause. I tell those patients who have to have the ovaries removed, as for instance in bilateral ovarian tumors in young women, that premature menopause will follow the operation, but that the climacterium is shorter, though it may be more intensive than one that comes at a normal time. In extremely young women, in the early twenties, reaction to the lost ovaries does not seem to create much disturbance. There will occasionally be a case that requires treatment, but many of these patients do not show reaction enough to bring them back for treatment. I explained that in my paper on the menopause some years ago as due to the relatively short duration of association on the part of the correlating glands.

By way of illustration I will cite 2 cases. A young woman twenty-three years of age was operated upon last July for bilateral ovarian cystadenoma, non-malignant. I never start these patients on treatment immediately. After operation I wait until I see what the reaction will be. She was operated in July, and on August 26th she told me she had begun to be bothered by flashes and sweats. She had gained in weight and felt perfectly well. This girl works for her living and hence must keep in good physical condition. I gave her extract of corpus luteum, 5 grains three times a day. By October 8th the hot flashes and sweats were much reduced, she had gone back to work, and felt very well. She then stopped the treatment for two weeks. On November 15th the hot flashes had returned, and she again resumed treatment. She showed some neuralgic pains and some flatulence at this time, which disappeared as soon as the extract was continued. She again stopped treatment in February, and then noticed a period of

depression or "blues," which she thought had a tendency to come on once every month. These had been variable, the last one of four hours' duration. On March 28th she reported that she had had no hot flashes or chilly sensations of any kind so long as she took the corpus luteum. This case also illustrates the point that treatment may be carried out quite satisfactorily with the dry extract in tablet or capsule form. The patient could not leave work every day or two for injections of extract in liquid form, and therefore used it in dry form. While the injection method is preferable, it is not absolutely essential.

Another patient is a woman of thirty-seven years, a professional violinist, upon whom bilateral oöphorectomy has been performed, also for benign neoplasm. If temperament enters into the situation, surely we ought to find it here. She told me there had existed always in her work an element of fear or diffidence when she appeared on the platform. Since the operation she has experienced a certainty of purpose she never had before. She has some hot flashes, some chilly sensations, but not enough to inconvenience her. She says, "I do not think I need treatment, unless you insist. I am perfectly satisfied and feel better than ever, particularly in my public appearance. I find I can appear with more control and more certainty than I ever hoped to achieve in my life."

Another patient upon whom I operated six months ago, a woman of forty-two, also had bilateral ovarian cysts. I told her to come back and let me know how she was in sixty days. I saw her in two months, and she stated that the reaction was not very marked and she felt very well. She was having hot flashes and chilly sensations, but she said unless they were more marked she could stand them very well. She came back this week, six months after operation, the hot flashes and sweats having increased, especially at night. I now have her under treatment.